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A STUDY OF ACHYLIA GASTRICA.

By Charles G. Stockton, M.D.,

PROFESSOR OF THE THEORY AND PRACTICE OF MEDICINE IN THE UNIVERSITY OF BUFFALO, BUFFALO, NEW YORK.

What is achylia gastrica? It was defined by Einhorn as comprising 'a class of cases in which there is absence of gastric secretion," and, "in which clinically the diagnosis of atrophy of the gastric mucosa seems to be justifiable." This writer would not include cases depending upon pernicious anemia. Apparently this definition describes a result following a variety of causes, hence we are led to conceive of the condition not as a disease, but as the result of diseases. If this be true, I fail to understand why the condition when occurring in pernicious anemia should not be included.

I have attempted to state here some conclusions reached in the study of 132 cases. Some of these did not fall strictly within the definition given. If, however, we are to form an opinion as to the causes leading to achylia gastrica, and of cases in the stage of development, then these cases should be included. I believe that it is wise to include them and to attempt the explanation of a pathological change that is not very uncommon.

The Incidence of Achylia Gastrica. These 132 cases are all private cases, not including hospital cases, and they bear a ratio to other stomach diseases examined as 1 is to 20. They may be divided into groups:

1. Cases in which there is persistent absence of secretion of ferments and of hydrochloric acid.

2. Cases in which there is a very low, but steadily decreasing secretion, which finally disappears entirely and remains permanently absent.

3. A group of cases which behave like one or the other of the preceding, save that at long intervals there may occasionally be found traces of combined chlorides or peptone.

4. There is rarely found a case in which all secretion is absent for a long period, after which there is a slow and gradual return (this group does not include the more frequently seen cases in which there is temporary absence of secretion, lasting for days or weeks, to be followed by normal digestion).

CAUSES. The various causes that result in achylia gastrica probably differ in nature: (*a*) Those that accompany pernicious anemia apparently result from a definite atrophy of glandular parenchyma of the stomach. (*b*) Others seem to follow gastritis. (*c*) Others appear to be secondary to general infection, possibly from gastritis, as is seen after typhoid fever, syphilis, etc. (*d*) There remains a large group in which, from unknown causes, the secretion becomes more and more depressed (as mentioned in the second heading) until complete achylia is established. It has been suggested that the trouble in the beginning is functional, and that subsequently gland structure disappears, similar to atrophy from lack of use in other regions. In attempting to follow the course of cases apparently about to become complete achylia gastrica, there is confessedly a source of possible error through misinterpretation. A case which shows a trace of combined chlorides or a faint biuret reaction may go on to complete loss of secretion; but, on the other hand, secretion may be found restored if the case is studied long enough. Nevertheless, these cases should be studied in relation to achylia gastrica, because in them only are we able to discover achylia gastrica in its process of development. To limit the discussion to cases in which no secretion has been found after prolonged observation is to limit the study not to the process itself, but to its result. Of course, there is profit in studying the effects upon the economy of the permanent loss of gastric secretion. This is one question, and it has value; but I feel that there is greater value in another question, that of analyzing the processes that lead to the permanent suppression of gastric secretion.

SEX. Of these 132 cases, 62 were males and 70 females.

AGE. Ages were from twenty-one to seventy-two years; but only five cases were under thirty and only one beyond seventy. There were 29 cases between the ages of thirty and forty; 37 cases between forty and fifty; 28 between fifty and sixty; and 25 between sixty and sixty-nine. A pretty even distribution between males and females occurred in these several decades.

OCCUPATION. In occupation the greatest variety occurred. There were 44 different occupations listed out of 130 cases. Appar-

ently occupation has no bearing. Perhaps it should be noted that 13 per cent. were farmers.

Previous Health. The previous condition of health is not as completely recorded in the earlier cases as I could wish. In studying the footings I am convinced that this is a matter of importance. In those in which the previous history was carefully recorded about 25 per cent. complained of long-continued stomach trouble; about 14 per cent. of prolonged mental strain or worry; 14 per cent. of influenza; and what seems to be of greatest significance, 20 per cent. had typhoid fever. In one case there is a definite statement that the stomach trouble began immediately after typhoid fever, that it never disappeared, and that complete achylia is now present. 10 per cent. had had pneumonia, and the history of hard drinking was found in only one case. There were only three cases in which syphilis was certain. One patient had a tape-worm and one had diabetes.

Of the accompanying diseases or morbid conditions, some stand out prominently. I am unable to state the proportion of patients subjected to a critical eye examination; but there were 30 recorded as having eyestrain. I believe that this condition was present in 50 per cent. of the cases. Pernicious anemia was present in 23 cases; marked arteriosclerosis in 8 cases; catarrhal gastritis in 6; hysteria in 4; persistent headache in 5; advanced nephritis in 3; marked tobacco intoxication in 2; marked alcoholic intoxication in 1; morphine habit in 2. Anemia was very commonly complained of.

Condition of the Intestine. Of 115 cases, the bowels were reported regular in 23; constipated or sluggish in 52; loose (diarrhœa) in 31; irregular in 9. The diarrhœa often followed a period of constipation. In connection with the state of the bowels, it seems to me important to consider specially the question of gastric motility.

In the majority of cases of achylia gastrica the stomach is found to empty itself more quickly than is normal. Frequently the test meal has to be withdrawn before the lapse of the usual time in order to find contents present. Occasionally the stomach empties itself almost immediately. This is found true in cases in which diarrhœa is present, and also when there is constipation. The diarrhœa was sometimes increased by nervous excitement, sometimes by unusual exercise. From long familiarity with these patients I have the fixed impression that overgastric motility is the rule, and that for a time there is present what might be called a compensatory constipation. This may remain or it may terminate in a diarrhœa, often postprandial in character, the act of eating seeming to excite an unusual motor activity which, beginning in the stomach, continues to the colon. Several times I have predicted the diagnosis merely from the hurried emptying of the bowel after eating. Especially is this true as regards breakfast. Lesions of the intestines are comparatively unimportant.

LIVER. In 33 cases the liver is recorded as diseased; in 23, large; in 6, small; induration in the region of the gall-bladder, in 2; tenderness around the gall-bladder, in 1.

KIDNEYS. The state of the urine was recorded in 80 cases, among which albumin was found in 33, tube casts in 16, overacidity in 19, indican in large amount in 13, alkalinity in 3, specific gravity relatively low in 21, amount decreased in 29, urea decreased in 40. This emphatically disposes of the statement once made that indican in the urine is the rule in low secretion of hydrochloric acid. The frequency in which albumin appears is somewhat remarkable. The lowered secretion both in the quantity of urine and in solids is interesting, but was to have been expected.

GASTRO-INTESTINAL SYMPTOMS. Vomiting was complained of in 29 cases, sometimes at rare intervals, often at periods of a few days only; sometimes excited by a hearty meal or by very cold or very stimulating food. Nausea was present in 10 cases, regurgitation in 11, sour regurgitation in 6, eructation in 24, pain in 19, sense of soreness in 9, anorexia in 20, hyperorexia a number of times, gastric distress in 30, excessive salivation in 2. I have been unable to find any relation between achylia and ptosis or gastrectasis.

Gastric symptoms are sometimes very conspicuous. One patient had excessive vomiting for two years, bringing up large quantities of watery mucus. She said she felt sure there were two places where food went down. In one place it passed on, in the other it stopped; this she vomited. She suffered immediately after eating, especially meat, but she liked and, indeed, craved it. One of the most conspicuous stomach symptoms is appetite, which the patient is afraid to satisfy because of distress thus induced.

MENTALITY. There is a field for a perverted mental attitude in patients who know that they suffer from this condition, but that is true of all dyspeptics. One patient regularly wakes between twelve and three with distress and nausea. Then she regurgitates once or twice a tablespoonful of fluid, feels better, and sleeps until morning.

GENERAL SYMPTOMS. Generally patients with achylia gastrica suffer from vague general symptoms, and, sometimes, at most, merely suspect the stomach. Complaint is made of unaccountable fatigue, occipital headache, myalgia, disturbed sleep, mental depression, arthralgia, loss of weight. Usually there is complaint about the intestines or liver. One interesting patient suffered from the stomach for years; five years ago he practised gastric lavage for three months. Following this he had severe fever for five weeks, probably typhoid. Two months later he began desquamating, and within a year had lost the nails of his hands and feet. Afterward his stomach symptoms were better until within the last seven months.

General weakness or depression was complained of in 33 cases, nervous excitement in 4 cases, nervous depression in 14, loss of weight in 24, arteriosclerosis in 24.

A biuret reaction was discovered sooner or later in 20 per cent. of the cases, although only in faint traces, except in two or three rare instances when real improvement occurred; rennin or rennet zymogen ultimately appeared in ten cases when formerly absent; free hydrochloric acid appeared ultimately in three cases, together with general improvement in secretion. Lactic acid was rarely present in large amount, but appeared at least in traces in 44 cases. More than a normal amount of mucus was present in 62 cases.

I have observed in several cases of complete achylia in women that the symptoms were all ameliorated and the general health improved during pregnancy and lactation. In one case there was persistent diarrhœa occurring after meals, accompanied with great distress, hunger which was cut short before the repast was completed, and a sense of general depression and fatigue. Objectively there was the appearance of poor health. All these disappeared during pregnancy, and the patient gained rapidly in weight and a good condition continued until she was delivered. This was repeated during three consecutive gestations, and at no time was there gastric secretion. In another case there was marked improvement during pregnancy which terminated by premature delivery. With this event the symptoms recurred and the gastric secretion remained absent.

It has been stated that gastric secretion recurred in some cases after a long period of treatment. The first case seemed typical except that it was accompanied by marked excess of mucus. In about a month's treatment the secretion returned sufficiently to show traces of free hydrochloric acid with biuret reaction. The case was probably secondary to gastritis.[1] A second case under observation for four years had complete absence of all evidence of secretion for one year, after which there was present a biuret reaction during about half of the time. Traces of combined chlorides were found. After three years free hydrochloric acid appeared and the digestion was in every way improved. This was a case of "lues in the innocent." The third case had been under treatment at intervals during two years and three months, at the end of which time he had a biuret reaction and free hydrochloric acid. The man subsequently gained very much in weight and general vigor. In this case also there was usually present too much mucus. I think these cases should all pass for instances of achylia gastrica. The general picture was quite unlike that of cases of subacidity which go on showing presence of emzymes and, from time to time, a faint trace of hydrochloric acid. I am not able to say that arteriosclerosis occurred in a greater proportion of cases than would be expected in the same number of other chronic diseases. It is noted as having been found very marked in 8 cases.

[1] Faber and Lange (Ztschr. für klin. Med., 1908, lxvi), reporting 56 cases, hold that in all cases of achylia gastrica we have to do with a gastritis or a degeneration of the secreting structure of the mucosa.

Stomach symptoms are not uniformly present in achylia gastrica. The proportion of cases in which this is true, I am unable to state. The symptoms are often inconspicuous, complaint being made merely of a sense of "goneness" or hunger appearing too soon after eating.

In the examination of stomach contents of supposedly healthy individuals, the gastric secretion has occasionally been found absent. Such cannot be counted as instances of achylia except after repeated examinations have been made. Anyone who has repeatedly examined many stomachs will be struck by the fact that those that promise to be achylia will show in a few days a normal or even an excessive secretion.

In some instances of achylia the stomach is resentful of all acids. It is at times difficult to administer hydrochloric acid even in small quantities to these patients; at other times this remedy gives distinct relief and seems to benefit the patient generally. It has seemed to me that the administration of pepsin has added to the comfort and well-being of these patients.

I am indebted to Dr. Myrtle L. Massey for the intelligent analysis of the cases upon which this paper is founded.

DUODENAL ULCER AND ITS TREATMENT.[1]

By Max Einhorn, M.D.,

PROFESSOR OF MEDICINE IN THE NEW YORK POSTGRADUATE MEDICAL SCHOOL, NEW YORK.

Formerly duodenal ulcer was looked upon as a rare disease, which could hardly be recognized during life. When chronic ulcers of the stomach began to be treated surgically, duodenal ulcers were often met with at the operation. It was shown that, in many cases in which the ulcer was believed to be in the stomach, it was found in the duodenum. Pagenstecher[2] justly laid stress upon the importance of surgical treatment of duodenal ulcer. In 1899 he reported several cases of ulcer of the duodenum, which were treated successfully by him by gastro-enterostomy. Moynihan[3] points out the difficulty of the diagnosis of duodenal ulcer, and says: "The symptoms of duodenal ulcer are chiefly characterized by their lack of ostentation. In more than half of the cases in which ulceration is found at the necropsy, symptoms have never been present. In 151 cases collected by Perry and Shaw, in which duodenal ulceration was found post-

[1] Read at a meeting of the American Gastro-enterological Association, Atlantic City, N. J., June 5, 1909.

[2] Die chirurgische Behandlung des Duodenalgeschwürs. Deutsche Zeitsch. f. Chir., 1899, p. 541.

[3] On Duodenal Ulcer and its Surgical Treatment, Lancet, 1901, p. 1656.

mortem, there were 91 in which there were no antecedent symptoms." Treatment should be medical. Surgical treatment is in order: first in perforation; second, when abscesses develop; third, when pain and hemorrhages occur persistently; and fourth, when stenosis associated with dilatation of the stomach develops. The same author, in conjunction with Robson,[4] expresses himself in a similar manner: "Duodenal ulcer very frequently fails to yield to general treatment, and from our experience of these cases we believe it is much more frequently the cause of fatal symptoms than is generally recognized. In hemorrhage from the stomach or duodenum, as shown by hematemesis or melena, where general treatment has been tried and failed, and where the bleeding is persisting, or recurring after brief intervals, operation is advisable with a view to find and to secure, if possible, the bleeding point or points, or of performing gastroenterostomy in order to obtain physiological rest, and thus to favor natural hemostasis."

Mayo[5] calls especial attention to the great frequency of duodenal ulcers. In his 193 cases operated on for ulceration, he found stomach ulcer in 60 patients, duodenal in 119, and both combined in 14. As contrasted with ulcer of the stomach, duodenal ulcer is found more frequently in men than in women; according to Mayo, men furnish 73 per cent., women 27 per cent.; according to Robson, 86 per cent. and 14 per cent. respectively. Mayo tries to explain this as follows: "The curve of the duodenum in men is usually higher than in women; that is, the first portion of the duodenum in men is nearly always ascending, while in women it is often transverse. It seems probable that, for mechanical reasons, the alkaline secretions of the liver and pancreas more readily neutralize the acid chyme in the upper duodenum in women than in men."

Robson[6] agrees with the great frequency of duodenal ulcers. He says: "Operative experience shows that in a large proportion of cases duodenal and gastric ulcers co-exist, and that the real proportion of duodenal to gastric ulcers is 1 to 2 or thereabout." Regarding treatment, Robson says: "While gastric ulcer is distinctly amenable to medical treatment in the earlier stages, it is questionable if, when once peptic duodenal ulcer has passed to the stage of being recognized by definite symptoms, it is ever really permanently cured by medical treatment alone." Referring to a material of 66 cases of duodenal ulcer operated on by himself without a single death, Robson tries to define its symptomatology. There is usually pain two to three hours after meals. It is due to the irritating effect of the acid on the open ulcer, and partly to spasm of the pylorus and to dragging on adhesions which are often present.

[4] Diseases of the Stomach and their Surgical Treatment, New York, 1904.

[5] The Contributions of Surgery to a Better Understanding of Gastric and Duodenal Ulcer, Annals of Surgery, 1907, p. 810.

[6] Duodenal Ulcer and its Treatment, Brit. Med. Jour., 1907, p. 248.

The following symptoms are more or less characteristic: Intermittency of the attacks; dilatation of the stomach during the exacerbations due to pyloric spasm; hyperchlorhydria; vomiting not frequent; hematemesis or melena; and perforation in 10 per cent. of the cases. Surgical treatment consists in excision of the ulcer or an infolding of the duodenal wall at the site of the disease, or gastroenterostomy. The latter forms the rule.

Deaver and Ashhurst[7] agree with Robson with reference to the symptomatology of duodenal ulcer; they place the frequency of perforation at 25 per cent. M. Jaffe[8] mentions two cases of duodenal ulcer with perforation. Both were operated upon, one recovering and the other dying. In neither of them were there any symptoms of ulceration; perforation was the first sign of the disease. There was rigidity of the right rectus muscle, very intense pain, and tympanites in the region of the liver. The ulcers are usually small, about the size of a quarter dollar.

Whereas all of the mentioned papers were written by surgeons, a few important contributions have been published lately by clinicians. Kuttner[9] describes two cases of duodenal ulcer in children in the first decennium, the one being four weeks old and the other four years. Both died of gastric and intestinal hemorrhages. Autopsy showed ulcers in the horizontal part of the duodenum. Kuttner advises in all cases, when medicinal treatment is inefficient, to think of surgical intervention. Pewsner[10] reports a case of duodenal ulcer with autopsy. During the disease there existed at first a digestive and later a continuous hypersecretion of gastric juice. There was no pain on pressure in the epigastrium. nor was there any connection between the taking of food and the pains. Before death a severe hemorrhage occurred. At the autopsy an ulcer, the size of a dollar, with thickened margins was found on the posterior wall of the duodenum. No signs were found microscopically of a neoplasm.

Until lately a positive diagnosis of duodenal ulcers could be made only on the operating or postmortem table, as the clinical symptoms were not sufficiently marked to indicate the condition with certainty. Since I[11] have used the thread test I believe the diagnosis of duodenal ulcer can be made clinically with some certainty. I therefore consider it advisable to describe all the cases of duodenal ulcer which I had occasion to observe during the last year and in which the thread test was employed.

Case I.—Nathan B., aged thirty-three years, has been suffering for about eight years from frequently recurring attacks of gastralgia

[7] Surgery of the Upper Abdomen, 1909, p. 200.

[8] Durchbruch von Magen- und Duodenalgeschwüren, Berl. klin. Woch., 1908, p. 346.

[9] Ueber das Vorkommen von Ulcus duodeni im ersten Decennium, Berl. klin. Woch., 1908, p. 2099.

[10] Ueber einen Falle von Ulcus duodeni, Arch. f. Verdauungskr., 1908, xiv, 645.

[11] A New Method of Recognizing Ulcers of the Upper Digestive Tract, Med. Record, 1909, p. 549.

about two to three hours after meals, and severe constipation. The attacks last one to two months and alternate with intervals of freedom from pain. In October, 1908, the patient vomited a large quantity of blood (half a quart). On November 25, 1908, he was examined by me. The thoracic organs were intact, the stomach was not dilated, palpation revealing no painful area. The stomach bucket showed one hour after test breakfast: HCl + diluted twelve times; in other words a marked hyperchlorhydria. On November 26, 1908, the duodenal bucket was allowed to remain in the digestive tract over night, and was removed early on the twenty-seventh in the fasting condition. The bucket was filled with a yellowish bile-like fluid; reaction alkaline. The thread was colored golden yellow from 53 to 73 cm. and reddish brown from 61 to 62 cm. A similar result was found with the duodenal bucket on December 1, 1908—a reddish brown coloration of the thread between 61 to 62 cm. The patient was given alkalies, and his condition improved.

Case II.—H. L., aged thirty-eight years, has suffered for ten years from attacks of pain occurring once or twice yearly in the region of the stomach, associated with nausea, sometimes with real vomiting, and lasting from several days to two weeks. The attack usually begins suddenly at midnight; the patient awakes and complains of nausea. During the period of attack considerable stomach symptoms are present, particularly two to three hours after meals, together with heart burn, frequent eructations, and the raising of water brash. The patient usually feels rather weak during the attack, and must generally remain abed the first few days. In the intervals complete euphoria exists. He never had jaundice. The stool is generally regular, rarely constipated. Physical examination showed cardioptosis, hepatoptosis, atony, and ptosis of the stomach. The stomach contents one hour after test breakfast showed: HCl +: total acidity = 85; no occult blood. Palpation of the abdomen showed only slight sensitiveness to pressure toward the right of the linea alba, in the region of the liver and below. The duodenal bucket was twice used, the patient retaining it over night. The first test on February 11, 1909, showed, on withdrawing the bucket, slight resistance for about 15 cm., then no resistance until the entrance of the œsophagus was reached. The bucket was filled with bile; reaction alkaline; steapsin present; at 66 cm. from the knot a trace of brownish coloration for about 1 mm. was visible. The second test was made February 21, 1909, with the following results: thread colored yellow at the bucket; thread distinctly reddish brown, from 59 to 62 cm. Contents: HCl trace; after neutralizing steapsin and trypsin were found. The patient was told to wear an abdominal supporting bandage, and was given alkalies and olive oil twice daily. He felt better and gained somewhat in weight.

Case III.—J. H. S., aged forty years, traveller, has had no syphilis; is a moderate user of alcohol and tobacco. He has always been

well until five years ago. He then began to suffer from attacks of indigestion, distress in the stomach, constipation, and wind, all of which would be relieved by a movement of the bowels or by carbonated sodium. About two years ago he had a hemorrhage from the bowel (the blood was tarry). This had been preceded about two weeks by weakness, distress, and malaise. He did not observe any special diet, and felt well for about one year, when he had a second hemorrhage (melena), which was accompanied by pain two inches above the navel rather than in the pit of the stomach, as in the former attack. The quantity of blood lost on several successive occasions at that time was not very large. He was then very careful as to his diet (raw eggs, milk, and bread and butter, no meat). He had pains about two to four hours after eating, never immediately after meals. He finally got well. About November 1, 1908, he had another attack, which lasted until I saw him (February 2, 1909.) He then had the pain again in the epigastrium and not over the navel. His pain was never constant, and was relieved temporarily by eating, but returns again in one to two hours. Bowels have always been constipated. The physical examination of the chest and abdominal organs did not reveal anything abnormal. Palpation of the gastric and epigastric regions was not painful. One hour after test breakfast the gastric contents showed: HCl +; acidity = 115; no occult blood. On February 4 the patient was given the duodenal bucket for the night; on February 5 the bucket was removed in the fasting condition of the patient. The thread length was 65 cm. It was brownish at the bucket and between 58 to 59 cm. The contents showed free HCl +; no bile. Rectal alimentation and then liquid diet were advised.

Case IV.—B. F. M., aged about fifty-three years, was seen on February 1, 1909; had ten years ago a very severe attack of indigestion, causing spasms of pain which lasted several days. After about a week or ten days he suddenly developed jaundice, which, however, disappeared in twenty-four hours. About two days after that he had a slight paralysis, which left him speechless for twelve hours, but he rallied, and after a few months was as well as ever. Two years later he had a very similar attack, without jaundice and with no other after-effect. Four years later he again had an attack of indigestion just as severe, but with none of the other ailments. For the past two years he has had trouble with his stomach, which seems to increase. He has colicky pains, which generally leave him with a feeling of exhaustion. They come and go at all times, but seem to occur mainly about two hours after meals and to get less when patient eats something. They start about four inches above the navel. They generally go all the way around to the kidney, and while never very severe and nothing like as severe as eight to ten years ago, still they are very annoying and disturb patient considerably. They interfere with his appetite, and patient has lost about ten

pounds in weight in the last six months, weighing now one hundred and eighty pounds. He has not smoked for ten years, although he was formerly an excessive smoker, from fifteen to twenty cigars a day. He is a very moderate drinker, however, sometimes not taking alcohol in any form for two or three months. He also gave up coffee and tea on account of the tannin contained in them, which he thought was injurious to him. Nothing abnormal was found by the physical examination. One hour after the test breakfast the gastric contents show HCl +; acidity = 105; no occult blood; no stagnation of food. The duodenal bucket was twice applied on February 3 and 5, 1909. The thread showed a reddish brown discoloration between 66 to 59 cm. on February 3, and similar discoloration at 55 to 61 cm. on February 5.

Case V.—Moses O. S. aged about thirty-four years; salesman; used to be a moderate drinker; never smoked; never had syphilis. In 1900 he was taken sick with diarrhœa, and had to go to the toilet six to seven times daily, usually after eating. He had a pressing down sensation in the rectum and intestines, as though he wanted to go to stool. He was treated off and on until 1902, when he came to me. After some treatment he improved and went out West. In 1905, however, the diarrhœa returned; about two to three times weekly he would have loose bowels. He has a beating sensation in the bowels, as though his heart was in his bowels. Then he has a dull aching pain in the hypogastrium, as though his food had not properly digested. He has an anguished feeling in the stomach as if a lot of hydrochloric acid were rolling around in it. This seems to gather there about two hours after he has eaten, and gives him distress. It lasts until he eats again. He frequently has a passage that looks like goat or sheep manure, little hard round lumps. He sleeps well and has a good appetite. The examination showed that the stomach extends to about a hand's width below the navel. The right kidney is slightly movable. One hour after the test breakfast the gastric contents show: HCl +; acidity = 80. The duodenal bucket was twice applied (January 26 and March 30, 1909). The first test showed a distinctly reddish brown discoloration of the thread between 61 to 66 cm. The second test, which was taken two months later, revealed a trace of brownish discoloration of the thread at 65 cm. The patient was treated with alkalies and was advised to wear an abdominal supporter. He has gained in weight ten pounds in the last two and one half months, and feels considerably better.

Case VI.—J. F. H.[12]

Epicrisis. In all six cases the diagnosis of duodenal ulcer was made by the thread test. In four the symptoms pointed to duodenal ulcer with some probability; in two, however, the symptoms were in no way characteristic. In one patient who was operated

[12] See Medical Record, April 3, 1909.

upon the ulcer was found in the duodenum, one inch beyond the pylorus, as diagnosticated. Regarding the frequency of duodenal ulcer, the surgeons (Mayo-Robson and others) maintain that the proportion of gastric to duodenal ulcer is as 3 to 2, and according to Mayo even as 2 to 3, whereas pathologists find at autopsies a proportion of 10 to 1. This seeming contradiction can be easily explained by the fact that the surgeons see only those cases in which an operation is necessary. Among these the ulcers at the pylorus or in its immediate vicinity, as they lead to disturbance of motility of the stomach, occupy the first place. Therefore, the proportion of duodenal ulcers must be high. Most gastric ulcers not situated at the pylorus are cured (cicatrization) without surgical intervention. These ulcers or cicatrices are then found only at autopsy.

According to my own experience gained with the thread test, the proportion of gastric to duodenal ulcers is about as 4 to 1; of course, the proportion must be still higher in favor of stomach ulcers, as those situated at the fundus cannot be demonstrated by means of this test.

Symptomatology. In all six patients a marked hyperchlorhydria was present. In Cases III and IV there also was a slight continuous hypersecretion of gastric juice. Slight peristaltic restlessness of the stomach was found in only one (Case VI). Pains are generally present two to three hours after meals, and are alleviated by the ingestion of food. Moynihan calls this symptom "hunger pain." In nearly all the patients, periods of suffering alternate with periods of perfect euphoria. Hemorrhages (hematemesis or melena) were found in two of the cases. Usually constipation is present, but a tendency to diarrhea was found in two. Pain on pressure was absent in nearly all the cases.

Diagnosis. The symptoms just enumerated (except hemorrhages) are all caused by hyperchlorhydria and frequently occur without the presence of duodenal ulcer. If, however, in association with the above symptoms hemorrhages in the digestive tract occur, the diagnosis of duodenal ulcer is justified—for the real gastric ulcers generally act differently: pains immediately after meals, pressure pains in the epigastrium, etc. The diagnosis appears to become more positive if the thread test shows a distinct blood spot at a distance of 58 to 66 cm. from the teeth.

Treatment.—The treatment should at first be medical. In the mild cases regulation of the diet (frequent meals, abstention from highly seasoned substances, acids, and too fatty foods), improving the general condition by means of iron, arsenic, cold sponging, good air, avoidance of bodily exercise, and the use of alkalies are sufficient to effect a considerable amelioration, if not a cure. In several cases olive oil (two tablespoonsful morning and evening) seemed to be of service.

In graver cases of duodenal ulcer (hemorrhages, severe pain, etc.) a strict ulcer cure with rest in bed and rectal alimentation, and afterward fluid diet, must be instituted. In these cases large doses of magnesia and bismuth are of benefit: calcined magnesia, 0.5 (8 grains) bismuth subnitrate, 2 (30 grains), in powders—one powder three times a day, a half hour before meals. If a strict rest cure has been unsuccessful, or if we have to deal with severe hemorrhages endangering life, and returning frequently, or if obstinate spasm of the pylorus occurs, associated with severe pains in the pyloric region and slight peristalic restlessness of the stomach, an operation (usually gastro-enterostomy) is indicated.

In duodenal ulcer the clinician must advise surgical treatment sooner than in gastric ulcer, as the former, through its complications (hemorrhages, perforations, stenosis of the pylorus), endangers life much more readily than the latter. Gastro-enterostomy in these cases is fortunately attended with good results. The ulcer will then soon heal, as the gastric juice does not longer flow over the ulcerated surface in the duodenum or irritate it. At all events, the dangers of hemorrhage, perforation, and pyloric stenosis are thereby prevented.

CLINICAL EXPERIMENTS WITH HOMOLOGOUS VACCINES IN THE TREATMENT OF SEPTIC ENDOCARDITIS AND PYEMIA.[1]

By W. Gilman Thompson, M.D.,
PROFESSOR OF MEDICINE IN THE CORNELL UNIVERSITY MEDICAL COLLEGE, NEW YORK.

Following the method of Sir Almroth E. Wright[2] of the injection of homologous or autochthonous vaccines, I have treated a series of cases of septic (streptococcal) endocarditis and pyemia with results which have again demonstrated its effectiveness, and afford strong hope of greater success when the technique shall have become better understood. Of the series herein reported, three cases of septic endocarditis were cured, with one of serious pyemia, and in several other cases of septic endocarditis there was clinical evidence that the septic process had been completely controlled, although subsequently death ensued from such complications as tuberculosis or pneumonia, which as yet are beyond the influence of vaccine treatment.

In several of the cases polyvalent vaccines were employed, but without benefit, before homologous vaccines could be obtained, which latter proved effective. In fact, in no case in which the homol-

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11, 1909.

[2] Trans. Harvey Society of New York, 1907.

ogous vaccines were used was there failure to produce some degree of reaction, such as a downward temperature movement and other indications of at least temporary betterment, and several patients who came under treatment after months of illness, in a condition in which a fatal issue seemed imminent, gave evidence of an arrested progress of the disease for many weeks.

Case I.—*Septic Endocarditis.* The patient, an Italian laborer, aged thirty-four years, had been ill for a month before he entered the Presbyterian Hospital. He became suddenly ill with rigors, prostration, and fever having a daily periodicity. On admission the heart was found enlarged an inch to the left, with a loud, rough, booming apical systolic murmur. A similar murmur appeared subsequently over the aortic valve. Numerous petechiæ were present, and there were subcrepitant rales at the base of both lungs. The murmurs increased in intensity, and three weeks before death the lower lobe of the left lung became consolidated and fluid was aspirated from the pleura. A pure streptococcus pyogenes aureus culture was twice obtained from the blood, being once associated with a large colon bacillus. The percentage of polynuclear cells in the blood was 83.5, and the leukocytes numbered 14,600. Although the patient died forty-four days after admission, his temperature (Chart I) illustrated strikingly the influence of increasing doses of the vaccine derived from the first blood culture, which caused a total fall of 11° (from 104.5° to 93.5° F.) Recovery was impossible from the fact that, in addition to the vegetative endocarditis observed at autopsy, there were present acute nephritis, chronic pulmonary tuberculosis, pleurisy, and infarct of the spleen. Examination of the heart showed fine vegetations on the free border of the left anterior aortic cusp, on the ventricular surface; the posterior cusp was retracted and thickened along the free border, and presented a large mulberry vegetation, 1 x 2 cm.; on the corresponding aortic surface of this cusp were two cul-de-sacs which extended into the vegetation. The other valves and the coronary arteries were normal.

The patient received four subcutaneous vaccinations at intervals of six days, containing in series, 50,000,000, 100,000,000, and two of 200,000,000 dead streptococci. For the ten days preceding the first vaccination the temperature had ranged steadily between 101.5° and 104.5° F. The first vaccination of 50,000,000 reduced the temperature in thirty-six hours from 104.5° to normal, and kept it normal and slightly subnormal for eighteen hours. It rose again to 103.5°, when the second vaccination brought it gradually down in two days to normal and subnormal. It rose again until it reached 105° F., when the third vaccination of double the previous strength again reduced it in thirty-six hours to normal, and finally subnormal, where it remained until the fourth vaccination.

Up to this time there appeared to be improvement in the patient's condition, commensurate with the decline in temperature. It was

determined, however, to give the fourth vaccination, but for the fourteen succeeding days before death the temperature remained markedly subnormal, and on one occasion fell as low as 93.5° F., as stated above. During this time the patient's pulmonary and pleural symptoms made serious advance, and he became rapidly weaker. It may have been an error to have given so large a final

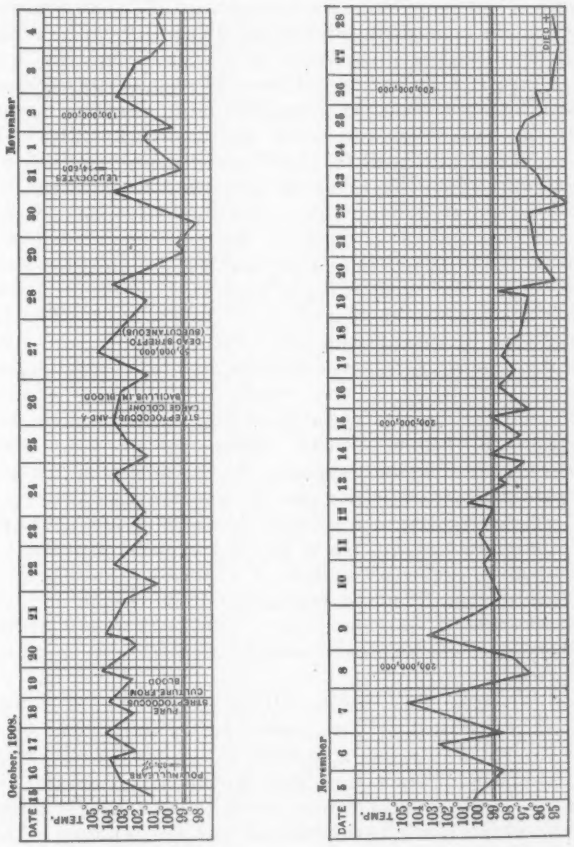


CHART I.—Streptococcic endocarditis, showing the effect of increasing doses of vaccine (Case I). Death from tuberculosis and nephritis.

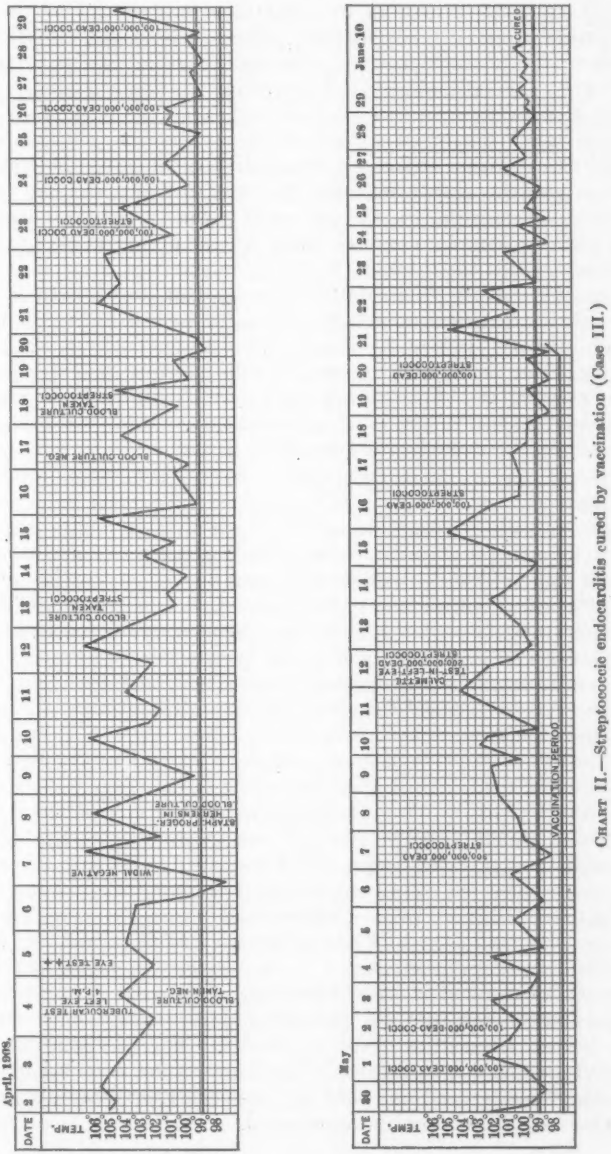
vaccination, but if so it was due to lack of experience with an entirely new method, although the probability remains that death was more directly caused by the severe nephritis, tuberculosis, and the extreme damage to the aortic cusp.

CASE II.—*Septic Endocarditis.* This was likewise an unfair test of any treatment, owing to the great number of complicating

maladies, yet it illustrates, in less striking manner, the influence of the vaccines on temperature. The patient, an Italian, aged forty years, had been at work in apparent health, until three weeks before admission to the Presbyterian Hospital, when he was attacked with paroxysmal rigors and fever. He felt somewhat better on alternate days, but was always feverish. One week before admission the calves of both legs became swollen and painful, presumably from neuritis, and palpitation was complained of. On admission there was no cardiac murmur, but later a loud aortic systolic murmur developed, and the apex was found in the fifth intercostal space 11.5 cm. to the left of the midsternal line. The leukocytes numbered 19,300, and the polynuclear percentage was 86.7. It is worth noting that as the case progressed and before treatment began, the leukocytes spontaneously dropped to 10,000 and the polynuclear count to 81 per cent. Two blood cultures gave pure streptococci. For some days there was a daily severe chill that accompanied great fluctuations in temperature, which on one occasion amounted to 9.5° F. (from 96° to 105.5° F.). Four weeks after admission a subcutaneous vaccination of 50,000,000 dead streptococci was given, followed in six days by another of twice that amount. The first vaccination kept the temperature at or below the normal for two days, and the second was followed by three days of normal temperature, when death ensued. There were so many lesions observed at autopsy that this case, like the preceding one, must have been doomed from the commencement. These lesions were: Senile emphysema with pulmonary gangrene; chronic interstitial nephritis and myocarditis; cholelithiasis; chronic pancreatitis; acute perisplenitis; early cirrhosis of the liver; abscesses of abdominal lymph nodes; white infarcts of the spleen, and red infarcts of the kidney. The aortic valves presented acute and subacute vegetative endocarditis, and smears from the vegetations gave streptococci. In this case the vaccines unquestionably controlled the temperature, but, owing doubtless to the numerous complications present, produced no other effect.

Case III.—*Septic Endocarditis.* The patient was a railway guard, aged thirty years. Two weeks before admission to the Bellevue Hospital he became acutely ill with a sore throat, chills, sweating, headache, slight cough, and mucous expectoration. On admission no cardiac murmur was heard, but a fortnight later a loud, harsh, systolic aortic murmur developed and persisted. During the first three weeks that the patient was under observation he exhibited an exceedingly irregular temperature (Chart II), which several times rose to 106° F., and on one occasion fluctuated 9° F. in one day (from 97° to 106° F.). During this period, also, five blood cultures were made, three of which gave pure stains of Streptococcus pyogenes aureus, but it is interesting to remark that the first and fourth were sterile. The leukocytes numbered 16,000, and the polynuclear

percentage was 84. During the succeeding four weeks ten subcutaneous vaccinations were given at varying intervals, some of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them on succeeding days, others at intervals of four or five days. In 8 of the vaccinations 100,000,000 dead streptococci were given, derived from the patient's own blood cultures. In one instance 300,000,000 and in another 200,000,000 were given. Thus, in all, the patient received 1,300,000,000.. The injections had remarkable effect upon the temperature, reducing it within two days from 105° F. to normal, and repeatedly reducing it when it tended to rise again. It never became so markedly subnormal as in the two preceding cases. There was coincident improvement in all the symptoms. Having been apparently dying, the patient gained in strength and weight, the leukocyte count dropped to 8000, and the polynuclear percentage to 53; the temperature finally remained normal and recovery became complete, with a gain in weight of thirteen pounds.

Case IV.—*Septic Endocarditis.* The patient, a girl, aged seventeen years, had had a septic temperature for more than five months, with occasional chills and sweating, when first seen by me in consultation. A loud, harsh, mitral systolic murmur had developed after a few weeks of illness, and petechiæ were present. Despite the apparent good digestion of ample food, emaciation was extreme, and reached a degree rarely equalled in the young excepting in protracted cerebrospinal meningitis. At my suggestion a blood culture was made by Dr. Hastings, and it proved to contain Streptococcus tenuans. This germ, injected in a mouse, caused death in three days, with extraordinary emaciation, so that the animal appeared to be merely skin and bones. A vaccine made from the blood culture was given repeatedly in doses of 13,000,000 to 20,000,000. The dosage was small because of the feebleness of the patient and the difficulty of giving hypodermic injections to one so emaciated. About two dozen vaccinations were given, at first one every other day, and later one every day. The effect on the temperature, doubtless owing to the small dosage, was less spectacular than in the three previous cases, but the vaccinations steadied the temperature, that is, converted it from a markedly septic type with wide daily excursions to a continued type with a gradual favorable downward trend. The heart action also became steadier and less tumultutous, the systole being better sustained. On one occasion the temperature fell to normal for the first time in a month after vaccination, and there was a coincident fall in the pulse rate from an average of 120 to 94. A blood culture made after several vaccinations had been given, showed a marked reduction in the number of germs present. It is conceivable that in this case the refractory temperature was in part due to tissue starvation, for I have rarely seen greater emaciation. Normal heat regulation in such a case naturally would be disturbed. The tendency to emaciation and asthenia, however, did not yield to the vaccination treatment, and after two months the patient succumbed.

CASE V.—*Septic Endocarditis.* The patient was a young woman, aged nineteen years, pregnant on admission to the Presbyterian Hospital. On admission she was very weak and emaciated, and she exhibited a septic temperature which ranged from 97° to 103.5° F., progressive anemia, petechiæ, and a loud, harsh, but variable systolic murmur over the base of the heart. Polynucleosis reached 89 per cent., but the leukocyte count was never high, the maximum being 10,300, with subsequent spontaneous fall to 6000. Two blood cultures were negative, and 5 injections of 10 c.c. each of Parke Davis' antistreptococcus polyvalent serum produced no effect of any kind. A third culture gave Streptococcus pyogenes aureus, from which a vaccine was made by Dr. Meakins. Two vaccinations of 100,000,000 each and one of 200,000,000 were given at intervals of four days. The effect on the temperature resembled that in the previous case, that is, the maximum was lowered a degree and the course became steadier and distinctly less septic. Shortly after the second vaccination the patient was delivered of a seven months' feeble infant, which lived about twenty-four hours. I was interested to learn whether this infant's blood was infected, but a culture proved sterile. Soon afterward the mother died, the vaccinations having proved of merely slight temporary benefit.

CASE VI.—*Septic Endocarditis.* The patient was a young married woman, aged twenty years, who entered Bellevue Hospital with a septic vaginal discharge two weeks after an abortion. She had a septic temperature ranging from 101° to 103° F., and was very feeble. There was a general neuritis involving both arms and legs. She was curetted and a vaccine was made from the uterine streptococci. Subsequently large abscesses were opened in the left ankle, left hand, and both elbows. A second vaccine was made from one of the abscesses. The patient developed a faint pulmonic systolic and a presystolic mitral murmur while under observation. A few petechiæ appeared. After several vaccinations were given, the patient showed decided improvement and streptococci disappeared from the pus in the last abscess. In all, eight vaccinations were given, in quantities varying from 45,000,000 to 150,000,000 dead streptococci. Four blood cultures proved negative. After five weeks of fever the temperature began to decline, and a week later it became normal and so remained, while the patient completely recovered excepting the persistence of the heart murmur.

This case may fairly rank as a septic endocarditis, for, despite the negative blood cultures, streptococci were obtained from the uterus and abscesses, and the heart murmurs and petechiæ developed under observation.

CASE VII.—*Septic Endocarditis. Recovery from Sepsis; Subsequent Death from Pneumonia.* For the following abstract of an additional case observed at the Lincoln Hospital I am indebted to

Dr. Walter L. Niles, who followed the case in the service of Dr. N. R. Norton:

The patient was a Greek boy, aged sixteen years, who, two weeks before admission to the hospital, was taken ill acutely with palpitation, dyspnœa, œdema of the feet, and lumbar pain. Under observation he developed a septic temperature which lasted three weeks until controlled by vaccine. There was a diastolic and systolic aortic murmur and a systolic mitral murmur. Pericarditis with effusion required aspiration. Blood culture revealed Staphylococcus aureus. There was a leukocytosis of 18,000 and a polynucleosis of 91 per cent. In all, 10 vaccinations with homologous vaccine were given in periods varying from one to three days, and doses varying from 100,000,000 to 725,000,000. The vaccinations produced remarkable response in lowering the temperature, the largest dose causing a fall in two days of 8° F. (from 103° to 95° F.).

After sixteen days of vaccination treatment, the temperature remained normal and subnormal for a month, and the notes of the case record "clinical picture entirely changed; patient eats well, sleeps well, feels well; wants to get up." The blood cultures became negative and the patient was evidently cured of the septicemia. Unfortunately, at the end of the month he acquired pneumonia and died.

Case VIII.—*Pyemia.* The patient was a railway employee, aged thirty years, with pyemia. Ten days before admission to the Presbyterian Hospital the patient burned off a "wart" on his right hand with caustic. The wound became infected with staphylococci and the hand greatly swollen. Bronchopneumonia ensued, for which the patient sought hospital relief. The bronchopneumonia ran the usual course, but defervescence was interrupted by a septic temperature which lasted three weeks, with a maximum rise to 105° F. and daily fluctuations of 3° F. (Chart III). The right hand and wrist had to be incised twice. Shortly after admission the left hand became the seat of purulent infection, and likewise was incised. On the fifteenth day septic arthritis of the right knee-joint developed, with extensive fluctuation. On the nineteenth day pyonephrosis was discovered, and on the twenty-seventh day a rapidly spreading diffuse abscess formed over the upper left side of the chest wall and shoulder, which soon contained a pint of pus. I invited one of the visiting surgeons to open it, but the patient was so extremely weak that the surgeon said death would probably occur within a few hours, and thought it useless to operate. I therefore directed my house physician to insert an aspirating needle and remove what pus he could. The next day vaccine treatment was begun, and such prompt improvement followed that the surgeon consented to open the shoulder abscess, and evacuated a dozen ounces of pus. The knee-joint had been previously aspirated. I was embarrassed in obtaining a vaccine for this patient, because the first blood culture was sterile. In all,

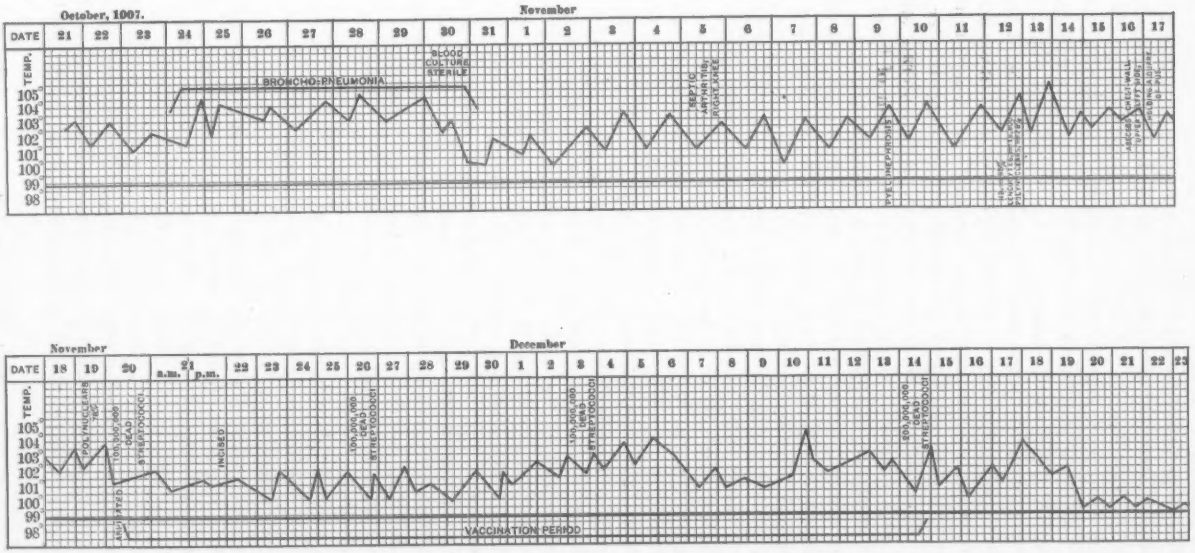

CHART III.—Pyemia; recovery after vaccinations (Case VIII).

six cultures were attempted. The third and fourth only showed Streptococcus pyogenes aureus, the four others being sterile. Four subcutaneous vaccinations were given at weekly intervals, three of 100,000,000 each and one of 200,000,000. Immediately after the first vaccination the temperature, which had averaged 103° to 103.5° F., fell 2°, and remained below 101.5° for three days, while the patient showed great improvement in every way. Subsequent slight elevations were controlled by the other injections until, after a total of two months of fever from the commencement of the illness, and one month from the first vaccination, the temperature remained normal, and recovery was complete excepting the damaged knee-joint. The patient finally left the institution with only moderate stiffness of the joint. The *x*-rays showed slight destruction of bone. Six months later I saw the patient digging a trench in the street.

In this patient the maximum leukocytosis was 18,800, with a polynucleosis of only 78 per cent., which latter subsequently rose to 87 per cent.

In another case of malignant endocarditis I was unable to obtain any vaccine, for four successive blood cultures were sterile, and there was no localization of pus. I therefore again employed in 10 c.c. doses the Parke-Davis polyvalent serum, made from virulent cultures of Streptococcus erysipelatus, but without benefit, and, in fact, without reaction of any kind. This patient was a youth, aged twenty years, who had a septic temperature ranging from subnormal to 105° F., anemia, emaciation, petechiæ, leukocytosis, varying between 45,000 and 20,000, and polynucleosis of 88.6 per cent. He developed a loud mitral systolic murmur while under observation, which autopsy showed was due to a circumscribed ulcer surrounded by a ring of vegetations and situated upon the lesser cusp of the mitral valve. The aortic valve was normal.

J. Barr[3] reports a case of malignant endocarditis with streptococcic blood infection, cured by vaccine treatment. Antistreptococcic serum was previously employed without effect, as in two of the cases above described.

There are certain diagnostic features observed in malignant endocarditis, illustrated by the cases previously cited, to which I would direct attention:

1. The observation of petechiæ is most important. While usually present first upon the legs, they may occur where they are easily overlooked. In three of my cases they were first seen on the lower eyelid, close to the sclerotic, and on the hard palate.

2. The ordinary blood count should not be too much relied upon in diagnosis, for it may prove misleading or variable, and may apparently improve as the patient grows worse. In one case of this series the leukocyte count fell spontaneously from 12,000 to 6000.

[3] Lancet, February 23, 1908.

I have records of several fatal cases in which the polynuclear count was below 60 per cent., and in one it was 53 per cent.

3. A negative blood culture should not prejudice against the diagnosis of septic endocarditis. In Case III herewith reported, two out of five cultures were sterile. In Case V, two out of three cultures were sterile, and in Case VIII, four out of six were sterile. In a fatal case, in which streptococci were obtained from vegetations upon the mitral valve, four blood cultures had previously proved sterile.

4. The character of the heart murmur is often distinctive. Usually most intense over the aortic region, it may be transmitted to the mitral area, or may be heard over the latter area first. It grows rapidly louder, and is of a harsh booming character when fully developed. It is usually accompanied by dilatation, and the pulse may be of the Corrigan type.

5. Anemia is progressive and extreme, and emaciation may become very pronounced, although digestion is seemingly unimpaired and the patient may take abundant food.

6. Peripheral neuritis with pain, tenderness, and sometimes swelling is not uncommon in the extremities, independent of abscess formation in the joints.

7. The temperature is characterized by extreme irregularity, and there are few conditions in which such wide daily fluctuations are encountered. In two of the cases above detailed, this variation amounted to more than 9° F. within half a day, and in another it was 8° F. On the other hand, the entire lack of septic temperature or petechiæ may lead to failure of diagnosis, as illustrated by the following case: The patient, a man, aged thirty years, entered the hospital nearly moribund from intense dyspnœa, cyanosis, and general anasarca. His heart was greatly hypertrophied and dilated, and there was a loud, harsh, mitral systolic murmur, with a fainter mitral presystolic, and an aortic stenotic murmur. The leukocytosis was 22,000. The case appeared to be primarily one of mitral regurgitation with intense chronic cyanosis, which had lasted several months. During the patient's life of three weeks in the hospital, the temperature never rose above 100°, nor did it vary in all more than a degree and a half. He died suddenly when he seemed to be improving, and the autopsy showed on the aortic cusp vegetations as large as a raspberry, with thickening and ulceration, and there were a few discrete pediculated vegetations on the larger cusp of the mitral valve, which yielded a pure streptococcus culture. Unfortunately no blood culture was made during life.

The safety and usefulness of the homologous vaccine treatment has been demonstrated by Wright, in whose original report[4] two cases of septic endocarditis were cited in which the temperature was markedly

[4] Loc. cit.

influenced, although the patients died. Much depends upon technique, the proper dosage, and intervals of dosage, as well as upon bringing the patient promptly under treatment before destructive lesions have developed in organs other than the heart. The difficulty of not always obtaining a positive blood culture from which to prepare an homologous vaccine may be overcome sometimes in other ways. In one of the cases of this series vaccines were made from streptococci derived from the patient's septic uterus, and subsequently from an abscess in the arm. In another case the vaccine was derived from pus in the knee-joint. The cultures and vaccines employed in the described cases were prepared for me by Prof. T. W. Hastings, of the Cornell University Medical School, and Dr. J. C. Meakins, of the pathological department of the Presbyterian Hospital, the vaccines being derived in each case strictly in accordance with the method originally devised by Wright.

As the subject is a new and important one, I have reported the fatal as well as the cured cases, with some degree of detail, for in several of the fatal cases the vaccines produced marked temporary beneficial effect, and the patient apparently was cured of sepsis, only to die of some other disease. In summary: Of the seven cases of septic endocarditis, three were cured by homologous vaccines; in two fatal cases the effect upon the septic phenomena was striking and temporarily beneficial, and in two fatal cases it was slight, but clearly demonstrable. In addition, one advanced case of pyemia was cured.

ACUTE GANGRENOUS PANCREATITIS.

By Louis Jurist, M.D.,
of Philadelphia.

The number of cases of gangrenous pancreatitis, especially of those eventuating in recovery, that have been reported, is so small that an additional case may be of value in bringing forward diagnostic and therapeutic points.

H. M. N., a male, aged thirty-nine years, a merchant, is an only child. His father died of some liver condition associated with ascites. His mother is living, but is a diabetic. The patient has had the ordinary diseases of childhood, typhoid fever when a boy, and pneumonia at the age of twenty-five years. He is married and has two children, living and well. He is temperate, fond of exercise, and eats moderately of plain food, but is occasionally costive.

Some nine years ago the patient first noticed a severe epigastric pain, lasting over night. After that, at long intervals, he had similar

pains, but of less marked severity. In the summer of 1907 he had a sudden attack of pain which had to be relieved by morphine, and since then the attacks have been somewhat more frequent. The last one was about six weeks before the present illness, and again morphine was used. In all of these attacks the paroxysm, while severe, has been of comparatively short duration and unaccompanied by jaundice.

On December 15, 1907, the patient again had severe abdominal pain during the night. He obtained relief from a small hypodermic injection of morphine, but for the next two days had more or less epigastric uneasiness and did not feel well. On December 16, when seen by me, he had some tenderness in the epigastrium, to the left and below the ensiform cartilage. On the following day the pain increased, but no morphine was required. Lavage of the stomach with about a quart of warm water appeared to give him relief. On the next day, December 18, 1907, the patient went to business, but still felt uncomfortable. About seven o'clock on the evening of this day he was seized with severe, agonizing pain in the epigastrium, and soon after this vomited.

Physical examination at this time showed the patient to be suffering severe pain, which was localized in the epigastrium to the left of the median line. A hypodermic injection of one-fourth grain of morphine, followed soon after by half that amount, gave only partial relief. The temperature was 95°, the pulse ranged from 80 to 90 per minute. The patient's skin had a sallow hue, he was perspiring freely, and his extremities were cold. He complained of slight epigastric tenderness and some nausea, and at times regurgitated a small amount of greenish fluid. Olive oil and calomel given during the night and next morning were also vomited. In the next two days the patient developed slight jaundice, which disappeared at the end of forty-eight hours. His bowels did not move until the second day. Elevation of temperature up to 100.2° was at times noted, and the pulse became rapid, weak, and irregular. The vomiting became less, but did not cease, and in the intervals the patient was troubled by persistant belching, which caused him great pain. Epigastric distention developed about December 20, and gradually became more marked. Associated with this was some slight rigidity of the left rectus muscle with epigastric tenderness. The gastric tympany extended to the navel, and the heart apex was pushed upward an interspace. About four days after the acute onset there was noted limited expansion of the left chest, some tenderness in the left loin, and a suggestion of a friction sound in the left axilla. The bowels were moved by enemas, but flatulence continued to distress the patient. The patient's general condition was one of marked toxemia, greatest at about December 21. His pulse was rapid and weak, the respirations were from 34 to 50 per minute, and the temperature irregularly febrile, but never exceeding

101°. The hemoglobin was low, at one time 56 per cent., while there was a continued and increasing leukocystosis, reaching a maximum of 23,400 on December 25. At no time, however, was there an excess of polymorphonuclear leukocytes.

The patient gradually improved as to his general condition. He was seen in consultation by Dr. John B. Deaver and Dr. John H. Musser, of Philadelphia, and on December 26, by Dr. Reginald Fitz, of Boston. At this time there was noted a deep-seated, not tender mass in the epigastric and upper umbilical regions, which was apparently continued into the left loin. There was marked tenderness in the left flank. The epigastrium was slightly distended, but there was no rigidity of the recti muscles. The edge of the spleen was palpable and there was an extended area of dulness in the left lower axillary region. The hepatic dulness extended from nipple to navel.

Up to this time there had been no laboratory findings of importance, except the leukocyte count at various times. The urine, at first scanty and high colored, later became more free. It showed albumin and a few casts, but no sugar. The Cammidge test was performed twice, once with positive and once with negative result.

The diagnosis of acute pancreatitis was first made December 21, and on December 26 all the signs pointed to an accompanying omental bursitis, without, however, an accompanying peritonitis. Operation was considered as being indicated, the only cause of delay being the hope of still further improvement in the patient's condition. This occurred in the next few days, and justified our postponement of operation.

The treatment medically up to the time of operation was comparatively simple. The patient was kept on very light liquid diet, and, as occasion required, was given small doses of strychnine. His bowels were kept open by enemas at first, but later these were not needed. At the time of greatest depression continuous saline enteroclysis by the Murphy method was given, and seemed to cause marked improvement both in the patient's general condition and in the kidney function.

On December 27, 1908, the patient was removed to the German hospital. On December 28, 1908, he was operated upon by Dr. John B. Deaver, under ether narcosis. The pancreatic collection was approached by the extraperitoneal route through the left loin, the incision being exactly as for a kidney operation. When the location of the pancreas was reached, thick dark necrotic material was evacuated. There was some fat necrosis of the fatty tissues along the line of entrance to the omental bursa. No attempt was made to do anything but drain the peripancreatic space and the omental bursa. This was done by means of two rubber tubes of large caliber; several gauze tampons were also introduced.

The cultures from the necrotic material removed at operation were

negative, as were also those from a slight amount of clear fluid aspirated into a hypodermic needle at operation, which seemed to come from just beneath the muscles and from a point above the necrotic and purulent material.

The patient left the operating room in good condition. For a week after the operation his pulse continued rapid, his respirations were above the normal, and the temperature showed marked fluctuations. It was often subnormal in the morning, and reached a maximum in the evening of 103.4°. The patient complained at times of chilly sensations and nausea.

On December 31 the leukocytes rose to 31,800, but with only 76.5 per cent. of polymorphonuclears and 15.5 per cent. of transitional cells. The lymphocytes, large and small, were but 6.5 per cent. On January 9 the left chest was aspirated because of the still persisting dulness there, but no fluid was found. On January 8 a large slough was removed from the wound 4 x 2 x 2 cm. in size; microscopic section showed it to be necrotic pancreas. The wound after this seemed to discharge less, but the discharge again became very free. Twelve days later a portion of gangrenous pancreas (confirmed microscopically) 6 x 3½ x 3 cm. was removed, and on the following day, perhaps because of trauma by the instruments, there was a profuse hemorrhage from the wound. The wound was tightly packed, and after that improved steadily, although still existing in the form of a small discharging sinus at the time of the patient's discharge from the hospital on March 12. The discharge from the wound was always very irritating to the skin, and analysis showed it to consist of pancreatic secretion and pus cells. Trypsin was present in it.

After the operation the patient's temperature was irregular for a time, but gradually dropped to the normal level. The treatment was such as would be given after any grave operation. Some difficulty was experienced in regulating his diet at first, but no digestants except a little pepsin were given. The urine during his stay in the hospital was at no time scanty, and showed no sugar, but always contained a few casts and a trace of albumin.

At the present time the patient is in perfect health. The sinus has permanently healed, after several temporary closures just after he left the hospital. He has had no glycosuria, has gained flesh, and digests all foods, although he has had several slight attacks of indigestion.

An examination of his metabolism by Dr. D. L. Edsall, of Philadelphia, shows it to be practically normal and very satisfactory, considering his recent disturbance. While taking a diet liberally supplied with fat, an average of 103.5 grams a day, he absorbed 93.8 per cent., good even for a normal person. He absorbed 90.1 per cent. of nitrogen, a little below the normal, and the stools contained 1.8 grams a day, which also showed rather poor absorption. He

excreted an average of 12.3 grams of nitrogen in the urine, hence with the 1.8 grams in the feces he lost 14.1 grams daily while taking 17.9 grams. Hence he retained a daily average of 3.8 grams, which he was evidently using in building tissue.

A case such as this furnishes many points of interest from a purely surgical point of view, but to the practitioner or clinician the etiology and diagnosis are of paramount importance. I shall, therefore, scarcely refer to the surgical aspects of the case, and confine my remarks to the clinical features of this particular case.

As to the etiology we must be somewhat uncertain. The patient was not of the obese and alcoholic type usually said to be associated with acute pancreatitis, although a review of the later literature shows this to be less often so than was formerly supposed. The repeated attacks of transient colicky pain, with slight evidences of indigestion and constipation at times present, render a diagnosis of biliary calculus probable, even in the absence of jaundice at any time. The attacks of pain might be considered as being due to pancreatic lithiasis, but in the absence of marked signs of disturbance in the pancreatic function this is untenable. Indeed, this diagnosis has so rarely been correctly made that we may well leave it entirely out of consideration. It is possible that cholelithiasis was the underlying cause of the acute pancreatic lesion. Its direct causation is open to question. It is undoubtedly an "acute" case, yet at operation there was no evidence that a large amount of blood had been lost, although some degenerated blood pigment, etc., was present. It seems likely, in view of the patient's previous good health, and of a few days of indefinite illness before the actual onset of ultra acute manifestations, that pancreatic inflammation preceded the hemorrhage. The two factors mentioned are the only ones upon which we can base such a supposition, because clinically such a distinction at the time of onset is, of course, impossible. On this assumption, then, the course was that of some biliary infection producing a mild inflammatory condition of the pancreas, and this in turn producing a hemorrhage. Following the hemorrhage a typical gangrenous pancreatitis occurred, such as would be expected after interference with the blood supply of a large portion of the organ. A patient in whom this is the sequence of events would seem to stand a somewhat better chance of recovery than one in whom the hemorrhage is primary, due to a diseased bloodvessel wall. In such case we have to deal not only with the grave local lesion, but also with the general arteriosclerosis, of which the pancreatic apoplexy is only a terminal manifestation.

The further course of the case as a gangrenous pancreatitis is to be distinguished from those in which a localized pancreatic infection causes a purulent condition of parts of the organ and its surrounding tissues. The former affection is undoubtedly the more grave.

The diagnosis in any case of acute pancreatitis is usually not easy. In this instance the rule holds good. At various stages of the disease during the first few days a number of conditions were simulated, at least to such a degree that a definite diagnosis was impossible. Before the acute paroxysmal onset of the graver symptoms the case presented no evidence of being anything more than perhaps an atypical form of gastritis or gastroduodenitis. The patient seemed slightly more ill than he should be under those conditions, but nothing definite supervened. At the time of the acute attack of pain, that is, the actual beginning of the gangrenous pancreatitis, there was nothing pathognomonic, yet the symptoms were suggestive. At that time they were: agonizing epigastric pain, subnormal temperature, slow full pulse, and absence of a localized point of tenderness or local abdominal signs.

The pain might be taken to resemble that of either a perforating peritonitis of the upper abdomen or an attack of gallstone colic. It was referred to the epigastrium and at times to the left thereof, but not with any great degree of definiteness. As against possible gallstone colic we have the absence of any radiation to the shoulders, back, etc. But it may safely be stated that at its very onset it was impossible to differentiate this pain from either of those mentioned. The subnormal temperature would occur in any severe sudden abdominal trauma, and depends upon the associated shock. Of more importance is the pulse. This in the attack was slow, regular, and of good volume. The association of a good slow pulse with subnormal temperature and shock is infrequent. Especially does it serve to eliminate from the diagnosis a perforative peritonitis, for in this the pulse practically always is rapid and often weak.

The association of slow pulse with acute pancreatitis has been dwelt upon by the later German investigators, notably Noetzel and Doberaner, both of whom have noted but have not attempted to explain the condition. Mayo Robson does not speak of it, however. John B. Deaver states that in his experience the presence of a slow pulse at the very onset of the attack is common. This must, however, not be confused with the pulse condition some hours later. In practically every instance of a true acute hemorrhagic or gangrenous pancreatitis the pulse in a very short time becomes rapid, weak, and often irregular, as in the present instance. Cyanosis, often included in the syndrome of an acute pancreatitis, was absent in this instance, as were also any definite localizing signs in the abdomen at the onset of the attack.

The diagnosis then at the onset was impossible, but within twenty-four hours certain added features tended to make a definite diagnosis possible. Obstinate constipation supervened, which could not be overcome by ordinary means. Vomiting, which had occurred once before the acute attack of pain, now took place several times in the twenty-four hours. Both of these conditions are practically

always present in acute pancreatitis. More important, however, as a subjective sign was the obstinate belching. Probably due to a diaphragmatic inflammatory involvement from the inflamed retroperitoneal space, it has been noted almost without fail in every case of acute pancreatitis. Not only this, but it has been to the patient a far more distressing thing than even severe vomiting, as each eructation seems to cause great pain.

Slight icterus came on within twenty-four hours, and remained forty-eight hours. This also would point to a pancreatic or biliary condition as opposed to a lesion of one of the upper abdominal viscera. In addition to these general symptoms occurring within the first forty-eight hours, certain other local signs, together with the change in the patient's general condition, tended to make a diagnosis of pancreatic trouble stand out alone as by far the most probable.

The general condition of the patient was one of profound toxemia rather than one of infection. With a rising leukocyte count and some fever, there were no marked exacerbations in the fever, chills, etc. At the same time the depression was so profound that there could be no mistake as to the toxic condition. The differential leukocyte count also showed a persistently low percentage of polynuclears, another point in favor of a toxic agent not purely bacterial in origin. And, indeed, recent investigations tend to show that in these cases there is a specific form of trypsin poisoning, which accounts for the constitutional condition that is so grave a feature of the disease. The local symptoms as described in the history of the case indicate, when considered in sequence, the gradual formation of an exudate deep in the upper abdomen and gravitating to the left loin.

Taken collectively, and regarded in the view of subsequent occurrences, the symptom syndrome in this case is fairly typical of acute pancreatitis. In the differential diagnosis of the case, however, we had to deal at the onset with a very acute condition, in which it was imperative to decide whether or not surgical interference was called for, and each day changed the aspect of affairs.

The differential diagnosis of acute pancreatitis under any circumstances is one of considerable difficulty. Cases have been reported in which intestinal obstruction, acute pelvic disease, acute appendicitis, etc., were simulated to a greater or less degree. I shall not attempt to enter into the general aspects of the matter, but merely to discuss it as applying to this partiticular instance. At the beginning of the illness in this case, or rather at the onset of the ultra-acute symptoms, three possibilities were to be considered. The pain was epigastric, everything pointed to an upper abdominal lesion. Therefore, acute pancreatitis, biliary colic, and the actual or impending perforation of a gastric or duodenal ulcer presented themselves. The pain resembled closely that of biliary colic, and

with the history of a previous attack of pain, a preceding digestive disturbance and the patient's sallow appearance made this the first thought. Against it we had the absence of any palpable condition about the gall-bladder and of any tenderness in this region or over Mayo Robson's point. The shock with slow pulse would have been a possibility in this condition also. The subsequent course of the illness soon showed a purely biliary condition to be out of the question. The toxemia and depression, beginning epigastric swelling, tympany, and tenderness, especially to the left of the median line, proved that we had to deal with a condition more grave than simple biliary colic, even one associated with marked inflammation of the bile passages. Nothing at any time pointed to a perforative lesion of the gall-bladder or ducts. Perforated gastric or duodenal ulcer would give the pain and shock. Yet in this we should have a rapidly spreading peritonitis of the greater cavity, with its typical symptoms, with rapid running pulse, etc., unless the perforation were partly walled off or had taken place upon the posterior wall of the stomach. This is unlikely, and in any case the symptoms would more probably have been those of a septic purulent condition in the greater or lesser peritoneal cavity. The possibility was borne in mind, however, until the toxemia with belching and icterus and the localizing signs deep in the left side left no doubt as to the true condition.

A word or two as to the laboratory findings may not be out of place. They were carefully considered, but, as in most cases they should be, they were counted entirely secondary to the clinical signs. The persistently high leukocyte count, with low polynuclear percentage, pointed to an intoxication rather than a pus infection. The comparatively high count, also, pointed to better powers of resistance than a low one would have done. The absence of glycosuria, except in one doubtful test, did not weigh against the diagnosis of acute pancreatitis, for, as Körte and others have pointed out, glycosuria is a rare accompaniment of the acute pancreatic lesions. The Cammidge test was, as stated, once positive and once negative, before operation. Until it is further developed, this must still be considered more as an interesting test than an important one, at least in the acute forms of pancreatic disease.

The treatment of acute pancreatitis is, of course, surgical in all instances. To the medical man such a case as this presents but one problem, surgically speaking—that is, when to advise operation. Recent German operators who have had opportunity of seeing a number of these cases advise early operation, to avoid the toxemia, to establish drainage, etc. Doubtless this may be the correct course to pursue in many instances, yet we must be guided by the condition present. In the present case, I have no doubt that immediate operation, say within forty-eight hours, when the depression and toxemia were at a maximum, would have resulted fatally, and that

by giving the patient a chance to react therefrom we gave him his one chance of recovery.

In conclusion, I wish to thank Dr. John B. Deaver and Dr. Reginald H. Fitz, of Boston, for their interest, and the use of their notes to supplement my own.

THE TYPHOID CUTANEOUS REACTION.

By Cleaveland Floyd, M.D.,
and
Williston W. Barker, M.D.,
of Boston.

In any disease, such as typhoid fever, in which the type of infection is that of a septicemia, every portion of the body is affected. This is true not only of tissue directly invaded by Bacillus typhosus, but on account of its diffusible toxins, of all the cells of the body. The dissemination of the endotoxins of the organism, through its destruction in the course of the disease, makes this possible, and when large numbers of bacilli are broken down by the action of bactericidal substances, the tissue cells become thoroughly sensitized to the poison. The rapidity of this sensitization is often marked, but it is frequently of short duration. This is accounted for partly by the relatively short period of the infection and possibly by inherent properties of the toxin itself. The union of the toxic molecule of the typhoid bacillus with the cell proteid is apparently unstable and easily broken up. In tuberculosis the converse is true. Infection is generally of long duration, and therefore the continued action of the tubercle toxin on tissue is much more lasting. Furthermore, its union with the cellular elements of the body is much closer and more stable. The old inactive lesions of tuberculosis respond to the stimulation of tuberculin for years after activity has apparently ceased. The addition of tuberculin to the conjunctiva or skin liberates locally toxic bodies, and by the unity with previously formed antibodies a reaction is produced. The reactive powers of the body to the toxin of tubercle slowly decrease with the subsidence of the lesion, but reactivity is quickly renewed when antibodies have previously been formed by the inoculation of tuberculin. In such instances the response to this stimulation is often delayed for several hours over the usual time required for a reaction.

In typhoid fever in which the infection is of relatively short duration, except occasionally in the gall-bladder, the result is different. In a previous paper it was shown that cellular sensitiveness as shown

by the ophthalmic test lasted only as long as the active stage of the disease. To determine if possible, if this sensitiveness affected the skin as well as the conjunctiva, and if so, whether it could be used diagnostically, has been the object of this work.

PREPARATION OF THE SOLUTION. Typhoid bacilli of high virulence were grown in pure culture on agar-agar for twenty-four hours, at 37° C. The growth was then washed off with normal saline solution and the emulsion incubated for four days. At the end of this period it was sterilized in a hot water bath at 60° C. for one-half hour, and then standardized after the method of Wright. The sterile emulsion was then centrifuged and the clear supernatant fluid used for inoculation. The solution used was considerably stronger than that used in the ophthalmic test, being the extract of one billion bacilli. In some instances centrifugalization was omitted and the whole emulsion used for inoculation, with the result of more marked reactions. A standard solution was used in all instances, and not an emulsion of the homologous bacillus in the individual case.

METHOD OF INOCULATION. In making the test, exactly the same technique was followed as in obtaining the cutaneous reaction in tuberculosis. After cleansing the arm with alcohol a drop of the solution was transferred to it by means of a sterile platinum loop, and this was then worked into the superficial layers of the skin by means of a platinum lancet. The inoculated material was then allowed to dry and a second puncture made below as a control. On the day following the inoculation there appeared about the point of scarification a moderate red areola ranging from 0.5 to 1 cm. in diameter. This reached its maximum in about twenty-four hours, and gradually diminished in intensity, disappearing at the end of forty-eight hours. The presence of vesicles and œdema, so often seen in the cutaneous tuberculin reaction, was noticeable by its absence. Constitutional symptoms were lacking in all cases. In a series of cases of typhoid fever treated by Richardson with bacterial residues well marked local reactions occurred in a number of cases, and persisted for some time. There were, however, general as well as local reactions in the cases treated.

CLINICAL RESULTS. In all there were 48 inoculations on 47 patients, of whom 30 were ill with typhoid fever, 2 with β-paratyphoid infection, 2 with unclassified fever (clinically not different from typhoid fever), 8 with tuberculosis, 1 with pneumonia, 1 with acute articular rheumatism, 1 with pernicious anemia, and 2 were apparently normal individuals. Of the typhoid fever cases, the reaction was positive 19 times, negative 9 times, and rendered unsatisfactory by the patient twice. One of the cases of unclassified fever responded to the test, although giving no other evidence of typhoid infection. Neither of the cases of paratyphoid fever gave any reaction whatever. With the exception of a slight reaction in the patient having pneumonia, all of the definite non-typhoid cases gave no re-

action. All of the positive reactions occurred during the active period of the disease, the single case which was inoculated twice giving a much more striking result at the first inoculation than the second. This result is of interest when we note that in tuberculosis the repetition of the cutaneous test when it has previously been positive gives a more active response at the second inoculation. The failure of the cases of β-paratyphoid infection to respond to the test may be due to the different character of the organism causing the infection.

TABLE.

Case.	Age.	Disease. Typhoid fever.	Days entrance.	Widal Positive Day of disease.	Blood culture Positive. Day of disease.	Cutaneous. Day of disease.	Character.	Time in hours.
1	7	Relapse	14	16	neg. (2)	20	Positive	48
2	21	Typhoid fever	5	5	..	17	Positive	48
3	7	Typhoid fever	14	16	..	25	Positive	48
4	13	Typhoid fever	14	17	neg. (10)	39	Positive	48
5	50	Typhoid fever	10	12	23	22	Positive	24
6	38	Relapse	7	7	7	10		
7	25	Typhoid fever	14	20	..	18		
8	31	Phlebitis—typhoid fever	28	31	..	90	Positive	24
9	27	Relapse	7	7	neg. (2)	60	Positive	24
10	17	Typhoid fever	8	8	..	17		
11	30	Typhoid fever	14	14	..	21		
12a	31	Typhoid fever	6	6	..	7	Positive	24
12b	31	Typhoid fever	6	6	..	35	Positive	
13	4	Typhoid fever	8	8	..	35		
14	17	Typhoid fever	14	16	..	30	Positive	24
15	47	Typhoid fever	10	17	..	30	Sl. positive	24
16	3	Typhoid fever	28	29	29	32		
17	19	Typhoid fever	14	20	..	34		
18	10	Relapse	8	8	..	13	Positive	24
19	27	Typhoid fever	4	6	..	10	Positive	24
20	6	Typhoid fever	6	6	..	9		
21	7	Typhoid fever	14	15	..	17	Positive	24
22	7	Typhoid fever	15	15	..	17	Unsatisfactory	
23	24	Typhoid fever	7	7	7	9	Positive	24
24	27	Typhoid fever	9	13	10	10		
25	38	Typhoid fever	14	16	..	24	Unsatisfactory	
26	16	Typhoid fever	15	15	..	16	Positive	24
27	14	Typhoid fever	9	11	..	22	Positive	24
28	37	Typhoid fever	9	13	9	13	Positive	24
29	6	Typhoid fever	14	14	..	25	Sl. positive	24
30	40	Typhoid fever	35	35	..	43		
31	25	Beta-paratyphoid	8	13	12	16		
32	21	Beta-paratyphoid	21	30	24	45		
33	34	Fever, cause (?)	8	4	2	9		
34	19	Fever, cause (?)	14	2	neg. (2)	14	Positive	24
35	39	Pneumonia	7	..	..	8	Sl. positive	24
36	28	Rheumatic fever						
37	51	Pernicious anemia						
38 to 45, tuberculosis								
46 and 47, normal individuals								

No attempt has been made to discover how early this reaction appears in the course of the disease, and the figures as to relative incidence of positive Widal reaction and blood culture are purely incidental to the description of the case and in no instance competitive.

The value of the reaction in diagnosis is problematical. The proportion of negative reactions in this small series is so great that little can be inferred from a positive or negative result in a given case.

The use of bacterial residues of greater toxic power than the bacillary extract might increase the percentage of positive reactions very materially. If this be true, the cutaneous reaction might be made of real value. In so far as the reaction has been obtained in typhoid fever, it is in harmony with similar observations made in cases of tuberculosis, and substantiates the theory of general tissue susceptibility to bacterial toxins in certain general infections.

We wish to express our indebtedness to the physicians of the Massachusetts General Hospital and the Children's Hospital for allowing these studies to be carried on in their wards.

SARCOMA OF THE STOMACH.

By Logan Clendening, M.D.,

ASSISTANT IN INTERNAL MEDICINE IN THE MEDICAL DEPARTMENT OF THE UNIVERSITY OF KANSAS, KANSAS CITY, MISSOURI.

The following are the notes of two cases of sarcoma of the stomach that have come to autopsy in the pathological laboratory of the University of Kansas.

Case I.—Case of Dr. James F. Trexler and Dr. Jacob Block. H. H., male, aged thirty-three years. His father is living, aged seventy-two years; his mother is living, aged sixty-one years; one brother is living, aged twenty-seven years; and one brother died, aged eighteen years, of "inflammation of the brain." The patient is a bartender; is married, and has one child. He had no serious illness until he was eighteen years of age, when he contracted specific urethritis, from which he never fully recovered.

In 1900 Dr. Trexler was called to see the patient, because of a very large gastric hemorrhage. The day following the hemorrhage the patient passed several tarry stools. Under treatment he recovered symptomatically, and was not seen until October, 1903, when he presented the following picture: He was a tall, thin man, strong and wiry. He complained of pain in the thorax. The heart showed an enlarged area of dulness with distinct heart sounds, but no murmurs (fluid in the pericardium). The pulse was 120. There was an area of complete dulness and absence of vocal fremitus and breath sounds over the lower lobe of the left lung. There was no pleural friction. The right lung was negative. A stomach analysis was never made. Urine (single specimen) was yellow, acid; specific gravity, 1025; contained no albumin or sugar. Microscopic examination was negative.

The patient died ten days later. The clinical diagnosis was pericarditis with effusion; left-sided pleurisy with effusion.

Autopsy Report. The body is that of a well-developed male, weighing 140 pounds. The body surface shows no evidence of disease.

The pupils are equal and dilated. The tissues of the chest are œdmatous, particularly on the left side and below the clavicle. The abdomen is tympanitic and contains a small amount of fluid. Both pleural cavities are full of greenish fluid. The pericardium measures six inches along the diaphragm. The sac is full of brownish fluid. After the lighter fluid is removed a quantity of free blood remains. The heart apex is displaced to the fourth interspace. The heart is small and is covered with a ragged, fibrinous exudate in the form of a membrane, which can be easily torn off. The auricles and ventricles are negative. Nothing of present interest is found in the lungs, the pancreas, the kidneys, the liver, or the spleen. The lymph nodes at the lesser curvature of the stomach are distinctly enlarged. The stomach contains a quantity of light chocolate-colored fluid. On the posterior wall near the lesser curvature, two inches from the pyloric ring, is an area three inches in diameter, whitish in color, presenting a slightly rugous surface, the entire area

FIG. 1.—Sarcoma of the stomach, showing as a round flat disk on the posterior wall. Two small pieces have been removed for microscopic examination (Case I).

somewhat elevated above the surface of the mucosa (Fig. 1). The growth extends outward in places, apparently following the rugæ. Over the stomach the veins are much dilated. The mucosa also shows a number of whitish follicles, one to two millimeters in diameter.

Microscopic Appearance of the Neoplasm in the Stomach. The tumor is a mixed-cell sarcoma, composed of round and spindle cells held in a loose, wide, network of fibrillary connective tissue. There are also numerous capillaries. The cells are small round, large round, and spindle-shaped, hematoxophile, with a small amount of cytoplasm and large granular nucleus. The nuclei at the edge of the growth next to the lumen of the stomach are large and vacuolated, and here the cells appear to be degenerating. The growth was evidently primary in the submucosa, invading the muscularis mucosæ and mucosa, leaving the muscle bands and serosa more or less intact. The mucosa is almost entirely obliterated in the circumscribed area of the growth, only a few tubules remaining, and these

at the periphery of the growth. The mucosa presents the thickest point of infiltration; the muscularis maintains its identity, but with considerable accumulation of cells between the muscle bundles. The submucosa is extensively infiltrated, but exhibits numerous strands of normal connective tissue intact. The visceral wall of the pericardium presents a metastasis of the growth. The blood-vessels in the vicinity are filled with sarcoma cells and the fibrous tissues are considerably infiltrated. The cells present in general the same appearances as in the primary tumor.

This case presents some points of considerable interest. Clinically, the great resemblance in symptoms to gastric ulcer is notable. In this instance, due to the fact that there was no tumor large enough to be palpated through the abdominal walls, the differential diagnosis must be considered absolutely impossible. Pathologically, the sharply circumscribed limitation of the tumor, the destruction of the mucosa, and the metastasis in the pericardium (the only record of such metastasis) are the interesting features. In the light of the autopsy findings we may reasonably believe that the first hemorrhage occurred from the sarcoma in the stomach, and that later metastasis formed in the pericardium, giving rise to an effusion three years later. The pleural effusion remains unexplained so far as this tumor is concerned.

Case II.—Dr. J. W. Perkins' case, St. Margaret's Hospital. W. H., aged fifty-eight years; male; December 7, 1905.

The patient had had no serious sickness up to six months ago, when he noticed loss of appetite and distress after eating. Later, he began vomiting once or twice daily, about two hours after eating. Sometimes he vomited immediately, "if he ate anything which did not agree with him." The pain after eating was relieved by frequent belching. He has lost about thirty pounds in four months. He is constipated; feels as though there were some stoppage below the stomach. He has had a cough for two or three months.

The patient is anemic, sallow, and cachectic. There is no jaundice. The tongue is large, moist, and flabby. A large mass, about the size of two fists, is felt in the upper abdomen in the region of the stomach, extending over the splenic region. The mass is movable and can be brought over to the right side. The heart and lungs are negative. The urine is negative. The gastric contents are not recorded. The clinical diagnosis was gastric tumor.

Operation disclosed a tumor of the stomach, involving almost the entire organ. The inoperability of the growth was apparent, and no effort was made to remove it. When the patient went home, twelve days after the operation, the tumor could be plainly seen through the abdominal wall. After death the stomach was sent to the laboratory for examination.

Pathological Report. The stomach is enormously enlarged; the walls greatly thickened, measuring at the pylorus two and one-

quarter inches. The pylorus, though surrounded by the growth, is patent. The neoplasm extends along the greater curvature, lesser curvature and posterior walls, as a lobulated tumor mass growing into the cavity of the stomach, especially at the pylorus (Fig. 2). The mucous membrane is generally smooth, but in some places in ulcerated. The serosa is smooth.

Microscopic Appearances. Mixed-cell sarcoma, with large and small round cells. The cells take the stain very unevenly, so that the tumor presents a varied picture to the eye: some cells have large some small dark staining nuclei; some large and some small light staining nuclei; some vacuolated or granular nuclei. There are a few spindle-shaped cells. The connective tissue groundwork is

FIG. 2.—The stomach opened, showing the sarcoma projecting into the lumen as a nodular mass (Case II).

composed of slender fibrils, and is quite abundant. There are numerous capillaries in the tumor substance. The growth is more strictly confined to the submucosa than in Case I, although the muscularis and mucosa are also involved. The tubules are not so widely destroyed as in the previous case, but the superficial epithelium of the stomach in almost entirely gone.

According to Howard,[1] the earliest report of a case of sarcoma of the stomach is by Bruch in 1847. Schlesinger,[2] in 1897, however, made the first comprehensive report, collecting 33 cases and report-

[1] Jour. Amer. Med. Assoc., 1902, xxxviii, 392. [2] Ztschr. f. klin. Med., 1897, xxxii.

ing three. Riegel's[3] article in Nothnagel's *System* is a review of Schlesinger's article. Brooks[4] in 1898, reported a case and analyzed 15 from the literature. Dock[5] reported a case in 1900 and collected 13 more. Howard, in 1902, in the most comprehensive article in the literature, adds 7 more cases from the literature and reports 4 of his own. The total number of cases reported is 61, which are carefully analyzed by Howard, whose digest of the literature is as follows:

Frequency. After carcinoma, the most frequent tumor of the stomach; 61 cases are reported.

Etiology. Sex: 30 cases in females; 25 in males. In 6 the sex is not mentioned.

Age. Youngest patient, three and one-half years (Finlayson[6]). Fifteen to twenty years, 6 cases; twenty to thirty years, 9 cases; thirty to forty years, 7 cases; forty to fifty years, 10 cases; fifty to sixty years, 10 cases; sixty to seventy years, 5 cases; seventy-eight years, 2 cases; over eighty years, 2 cases. Incidence in second half of life distinct.

Seat of Tumors. Diffuse, 13 cases; pylorus, 8 cases; pylorus and lower half of stomach, 6 cases; pylorus and greater curvature, 1 case; total, involving pylorus, 16 cases, producing stenosis in 5; greater curvature, 7 cases; greater curvature and posterior wall, 1 case; lesser curvature, 2 cases; anterior wall, 1 case; lesser curvature alone, 5 cases; cardia and lesser curvature, 3 cases; posterior wall and lesser curvature, 1 case; greater curvature and posterior wall, 1 case; posterior wall alone, 6 cases; seat not mentioned, 5 cases.

Size of Tumors. Pigeon's egg to man's head. (Baldy's[7] must have been larger, as he reports this remarkable tumor as filling the abdomen from the symphysis to the ensiform.)

General Appearance. Nodular in 27 cases; ulcerated in 10 cases. Tumors usually in the submucosa. Cystic, hyaline, calcareous, and myxomatous degeneration occurred. Metastasis occurred in 40 per cent; not so widespread as in carcinoma. Mesenteric glands, omentum, liver, spleen, and kidenys were seats of metastasis. (In my first case, the pericardium.)

Histology of the Tumors. Round cell, 16; spindle cell, 8; mixed cell, 4; lymphosarcoma, 15; myxosarcoma, 4; fibrosarcoma, 3; angiosarcoma, 3; endothelioma, 1.

Since Howard's article there have been eight articles describing this condition and ten cases have been reported. Oberst,[8] Dobromyslow,[9] Weinberg,[10] McCormick and Welsh,[11] each describe 1 case; Yates[12] reports 2 cases, Manges,[13] 3, and Dalton,[14] 1. Weinberg's case was a tumor arising from the lesser curvature and cardia with

[3] Nothnagel's Spec. Path., Band xvi.
[4] Med. News, 1898, i, 617.
[5] Jour. Amer. Med. Assoc., 1900, xxxv, 156.
[6] Brit. Med. Jour., 1899, ii, 1535.
[7] Jour. Amer. Med. Assoc., 1898, i, 617.
[8] Beitr. z. klin. Chir., xlv, No. 2.
[9] Russki. Chir. Archiv, 1902, Heft 5.
[10] Inaug. Dis. München., 1901.
[11] Scottish Med. Surg. Jour., 1906.
[12] Ann. Surg., 1906, xliv, 599.
[13] Mt. Sinai Hosp. Reports, 1907, 68.
[14] Trans. Lond. Path. Soc., 1906; Brit. Med. Jour., 1906, 1638.

metastases in the peritoneum and abdominal organs. Dobromylow's case, in a patient aged fifty-three years, was a spindle-cell sarcoma on the lesser curvature of the stomach near the pylorus. It involved the muscularis and left the mucosa intact. In the centre was a focus of softening.

Oberst's case was in a man, aged twenty-five years, who became sick suddenly, with hematemesis, pain in the epigastrium, blood in the stools, and great tenderness in the epigastrium, followed by a high, remittent fever and right-sided pleurisy. With expectant treatment, the bleeding subsided, but the infiltration in the epigastrium remained. The autopsy revealed a tumor, including the pylorus, sharply marked off from the duodenum, spreading out over the stomach wall and extending some centimeters into the liver substance. The tumor was fixed behind by strong adhesions. The stomach wall was 6 cm. thick. The atrophic mucosa covered the whole tumor, except in one place, where there was a deep depression the size of a five-mark piece, the bottom of which was formed by degenerated liver tissue. There was no lymphatic enlargement nearby. Microscopically, the tumor proved to be a round-cell sarcoma, originating in the muscularis, involving the mucosa, but not obliterating the glands and not involving the serosa. Oberst discusses the differential diagnosis between sarcoma and carcinoma of the stomach, and says that bleeding is less frequent in sarcoma. Metastasis, he states, is not always conclusive, as von Leube found a carcinoma of the stomach with sarcomas of the skin.

Norman Dalton's case occurred in a male, aged fifteen years. The symptoms were abdominal swelling and pain, covering a period of three or four months. At autopsy there was found a small round-cell sarcoma of the stomach and lymph glands.

Manges reports 3 cases: One was in a man, aged forty-eight years, in whom an epigastric tumor had developed between two examinations, four months apart. At autopsy a lymphosarcoma was found growing at the pylorus with metastasis in the pleura. One was in a woman, aged forty-eight years, in which the autopsy revealed a scar of an old ulcer at the pylorus and a soft sessile tumor, a myosarcoma, on the outer wall of the stomach near the pylorus. One was in a girl, aged nineteen years, who had bloody vomiting, pain after eating, and tenderness and pain in the left hypochondrium. At operation an inoperable diffuse lymphosarcoma was found involving almost the entire stomach.

Manges comments on the difficulty of diagnosis, due to lack of gastric symptoms. He calls attention to these points: (1) Gastric sarcomas arise from the muscularis and submucosa. (2) The growth may be diffuse (lymphosarcoma) or polypoid (spindle-cell). (3) The pylorus is rarely stenosed. (4) Degenerations frequently occur. (5) The tumors are often large. (6) The tumors may be primary or secondary. (He states that they may be local

manifestations of Hodgkin's disease, a statement which, in view of the recent studies of Longcope and Reed on the histology of Hodgkin's disease, seems unwarranted.) (7) The great range in the age of the patient.

Of Yates' two cases (operated upon by Dr. A. J. Ochsner), the first was a large round-cell sarcoma, which involved the greater curvature of the stomach, 3 cm. from the pylorus. It occurred in a woman, aged thirty-seven years. The second case is remarkable as being one of the few cases of cure on record. The patient was a man, aged forty-four years, who had had an attack of hematemesis four months before operation; later a tumor could be felt beneath the costal margin. It proved to be a large spindle-cell sarcoma, which arose by a pedicle from the posterior wall of the stomach. It was removed, and the patient, when examined seventeen months after the operation, was well.

Sarcoma of the stomach is of considerable interest pathologically, as in frequency it is the second among the tumors of that organ. From my study of the 2 cases and the descriptions of various authors, the usual type seems a mixed-cell sarcoma, composed of round and spindle cells lying in a connective tissue matrix of fine interlacing fibrils. From this group must be separated 3 cases, described as angiosarcoma, and 1 case of endothelioma. The tumors are generally reported as not involving the mucosa, although my cases are decidedly at variance with this. The tumors grow to considerable size, but seem not so disposed to metastasis as carcinoma (metastasis in about 40 per cent. of the cases). Sarcoma is of interest, too, as occurring in the majority of cases in individuals over forty years of age.

Clinically, it is of somewhat less interest. In three cases (Oberst's, Manges' third case, and my second case) the vomiting of blood in a young individual made the consideration of a differential diagnosis between the affection and ulcer important. The chemical analysis of the gastric contents in these cases gives us about as little help in diagnosis as such analysis ordinarily does. The total acidity runs about 50; the HCl is sometimes, though not uniformly, absent, and may be high. Sediment is modified by bleeding and presence of stenosis. The differential diagnosis between sarcoma and carcinoma is very difficult, and is unimportant as regards treatment. Pyloric stenosis occurs in about 7 per cent. of the cases. Fifteen cases have been operated upon. Dock's patient was well at the end of five months, and in 1 case reported by Yates the patient was well at the end of seventeen months. Yates also quotes Corner and Bairbank[15] as reporting 4 cases well at the end of four, five, twelve, and twenty-four months after operation. The tumor is usually fatal within one year.

[15] Practitioner, 1904, lxii, 810.

ENDEMIC AMŒBIC DYSENTERY IN NEW YORK, WITH A REVIEW OF ITS DISTRIBUTION IN NORTH AMERICA.

By Henry S. Patterson, M.D.,

ASSOCIATE IN APPLIED THERAPEUTICS IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK; DEPUTY ATTENDING PHYSICIAN TO THE HUDSON STREET HOSPITAL, NEW YORK.

The infrequency with which endemic amœbic dysentery is met in New York, and the prevailing belief that it is a disease of the tropics justify recording the following cases. Only the essential facts of the histories are included:

Case I.—S. W. McG., admitted to the Vanderbilt Clinic July 7, 1908; history 39,054; aged nineteen years; U. S.; single; electrician.

In September, 1907, the patient was suddenly taken with a cutting pain about the navel, intermittent, and lasting four days, at the end of which he began to pass "brown slime" which "looked like tobacco juice." At first he had as many as seven passages at night, accompanied by pain in the abdomen and rectal tenesmus. The diarrhœa kept up with variable intensity until a week before admission, when the symptoms became so much worse that he had to come to the clinic for relief. He had lost twenty pounds. At one time during his illness he was admitted to a hospital, but a stool was never examined. *He had not been off Manhattan Island since one day in the first two weeks of June*, 1907. On admission, the temperature was 98.2°; pulse, 92; respirations, 24; and the weight 112 pounds. He was poorly nourished and wasted. The tongue was moist and slightly coated. There was no lymphatic enlargement.

The heart was negative. The pulse was of good size and force. Physical examination otherwise was negative.

On July 8 a fresh stool was examined, and found to contain bodies like amœbæ, but no motility was seen. The proctoscope was passed and some bloody mucus obtained, which contained many actively motile amœbæ.

Case II.—W. K., admitted January 20, 1909; history 61,986; aged twenty-eight years; married; longshoreman.

Four years ago he began to have bloody diarrhœa, with about two stools at night and three in the day. For the last two years the diarrhœa has been more severe, with four to five stools in the day time, and an equal number at night. He has lost considerable strength, but no weight. No other symptoms. On admission he was well nourished, but pale. No abnormal physical signs were found. By the proctoscope bloody mucus containing a few motile amœbæ and many similar inactive bodies, was obtained.

On January 21, mucus obtained through the proctoscope and examined on a warm stage showed a fair number of motile amœbæ.

The patient states that he was born, brought up, and always

lived in Fishkill up to two years ago. (Fishkill is on the east bank of the Hudson, about sixty-six miles from New York.) He has never been outside of New York State except to unload bricks in New Jersey opposite New York City. For about seven years previous to his illness he had been working on a brick boat, plying between New York and Fishkill. The drinking water was at times taken from the river near Fishkill, when the water, after a rain, was not brackish, and allowed to stand in a barrel.

CASE III.—Clinic number 76,899; male, aged thirty-eight years; Russian; married; calciminer; admitted to the Vanderbilt Clinic May 19, 1909.

The family history and the past history are unimportant.

The patient came to America six years ago, and since his arrival has lived continuously in Brooklyn. In August, 1908, he developed dysentery, and was in a Brooklyn hospital for four months. He now has six or seven watery and bloody stools a day. Physical examination was negative.

On May 21, 1909, sigmoidoscopic examination showed small, superficial ulcerations; one just inside the sphincter was sloughing. Mucus examined microscopically on a warm stage showed fairly numerous motile amœbæ coli.

In the first case the patient was undoubtedly infected in New York City. Musgrave and Clegg,[1] whose admirable work of growing the amœba in symbiosis with bacteria harmless to animals and man marks an epoch in our knowledge of amœbiasis, found that in animals fed on cultures the period of incubation was from twenty to seventy days. Injections produced focal lesions containing amœbæ as early as the second day. To a healthy man, who had been observed ten days, three gelatin capsules were fed of three week's old cultures of amœba in symbiosis with a bacterium proved harmless to man and animals. On the twelfth day there was diarrhœa, and amœbæ for the first time were present in the stools. Consequently all the experimental evidence that Musgrave and Clegg have furnished would indicate that our first case was infected some time subsequent to his outing from the city.

Among New York physicians the belief is current that intestinal amœbiasis is to be expected only in persons recently returned from the tropics. Although an ill-defined impression exists in the minds of a few practitioners that occasional endemic cases of amœbic dysentery occur in New York, the existence of this condition is not as generally known as it should be, because the cases have been few and because systematic collection of them has never been presented to the profession. The important features of amœbic dysentery are not sufficiently sharply defined from those of other forms of colitis to enable one unacquainted with it to recognize the condition.

[1] Bull. 18, Bureau of Gov. Lab., Manila, p. 1.

Consequently the only way the diagnosis can be made in the majority of endemic cases is to suspect that the condition exists.

The following collection of endemic cases in New York City is offered in the hope that it will stimulate efforts to recognize the disease and determine its exact frequency in this vicinity.

In 1893 Brannon[2] reported the case of an inhabitant of New York who had been a sailor in his youth, but had given up that occupation years before his illness began, to become a grocer. There is nothing in the history to indicate that he was infected outside of New York. In 1886 he had a liver abscess, and in the following six years he had a succession of five other such abscesses, to the last of which he succumbed in March, 1892. Amœbæ were found in the liver and colon postmortem.

At a meeting of the New York Academy of Medicine, in 1900, Graser[3] reported two endemic cases. One patient was a hotel keeper, aged thirty years, who had never been out of New York. The other patient had never been farther than Staten Island. At the same meeting, in commenting on a series of twelve cases in his own experience, Brooks[4] stated that two of them were certainly infected in New York. In the discussion of a paper by Dock,[5] in 1902, Libman said that within the last year at the Mount Sinai Hospital specimens from at least ten cases of amœbic abscess of the liver had been examined, from which no history of any residence outside of New York City could be obtained.

Distribution Throughout North America. *Canada.* The late Dr. A. Brayton Ball[6] reported a case in 1892 that originated in Winnipeg. This was the first case to be reported from New York City.

Maine. Lockwood's[7] case in all probability contracted the disease in Old Orchard Beach.

New Hampshire. One of the earliest American cases on record was that of Gerry and Fitz,[8] who, in 1891, contributed the history of a young woman, who was supposed to have contracted the disease in the White Mountains.

New York. Stockton[9] reported, in 1894, an endemic case from Buffalo. He mentions a case occurring in his practice the preceding year. Nydegger[10] mentions a case occurring in the practice of a medical friend in the northern part of the State.

Pennsylvania. Musser[11] was among the first to report endemic cases. In December, 1890, he reported from Philadelphia two

[2] New York Medical Journal, 1893, lvii, 317.
[3] Medical News, 1900, lxxvi, 433.
[4] Medical News, 1900, lxxvi, 433
[5] Jour. Amer. Med. Assoc., 1902, xxxix, 617.
[6] Therap. Gazette, 1892, 3 S. viii, 523.
[7] Medical Record, 1897, li, 475.
[8] Boston Medical and Surgical Journal, 1891, cxxv, 592.
[9] Internat. Clin., 4 S. i, 69.
[10] West Virigina Medical Journal, 1907, ii, 11.
[11] University Medical Magazine, 1890, iii, 116.

hospital cases, and mentioned two occurring in private practice. About the same time Stengel[12] reported three, one of which was a private case alluded to by Musser. A few years later Musser and Willard[13] reported another case from Philadelphia.

Maryland. In 1890 Lafleur[14] reported the first endemic case in this country. Since then Howard,[15] Street,[16] Preston and Rurah,[17] Lewis,[18] Amberg,[19] and Boggs,[20] have reported a large number of cases.

District of Columbia. In a series of cases treated at the Johns Hopkins Hospital, Boggs reported three from the District.

Virginia. In the series just alluded to, Boggs reported fourteen from this state. Slaughter[21] has reported a case probably infected in Richmond.

West Virginia. In the series reported by Boggs, eight acquired the disease in this State.

North Carolina contributed five cases to Bogg's series.

South Carolina. Wasdin[22] has reported three cases.

Georgia. Boggs has contributed four cases, Wasdin one, Harris[23] thirty-two, and Diamond[24] three.

Florida. Boggs has contributed two cases. I myself have seen two cases which were infected in this State.

Tennessee. Boggs' report includes one case from this State, while Harris has reported two.

Alabama. Boggs has reported four cases, Harris and Withington[25] one each, while Taylor[26] has reported a fatal case from the centre of the State.

Mississippi. McElroy[27] reports a case from Stovall.

Ohio. Eichberg,[28] in 1891, reported from Cincinnati a case of amœbic liver abscess, no statement being made that the case was imported.

Illinois. Kurtz[29] has contributed a case from Neoga, while Preble[30] from Chicago, reports two cases; the first patient had lived for nine years in the city, and had been in the country a few weeks, six months previous to the beginning of the illness. The second patient had not been out of Chicago for thirty years.

[12] Medical News, 1890, lvii, 500.
[13] University Medical Magazine, 1892, v, 525.
[14] Johns Hopkins Hospital Bulletin, 1890, i, 91.
[15] Medical News, 1892, lxi, 705.
[16] Maryland Medical Journal, 1893-94, xxx, 92.
[17] New York Medical Journal, 1894, lx, 593.
[18] Maryland Medical Journal, 1896, xxxv, 145.
[19] Johns Hopkins Hospital Bulletin, 1901, xii, 355.
[20] Virginia Medical Semimonthly, 1908, xiii, 9.
[21] Virginia Medical Monthly, 1895–96, xxii, 722.
[22] Medical News, 1891, lix, 656.
[23] Amer. Jour. Med. Sci., 1898, N. S., cxv, 384.
[24] Phila. Med. Jour., 1900, v, 817.
[25] Boston Medical and Surgical Journal, 1894, cxxx, 516.
[26] Alabama Medical Journal, 1903–04, xvi, 10.
[27] Memphis Medical Monthly, 1902, xxii, 169.
[28] Medical News, 1891, lix, 201.
[29] Medicine, 1903, ix, 595.
[30] Chicago Medical Recorder, 1899, xvi, 33.

Missouri. Nietert[31] has reported seven cases which developed in or near St. Louis. Meyer[32] observed a case in St. Louis and mentioned one of Fischel's, while Funkhouser[33] has seen one originating in the same city. Murphy[34] has contributed a case from Kansas City.

Michigan. Dock, in 1902, reported one case infected in Michigan. The patient had not been out of the State since 1893. There is no history of any contamination by persons returning from the tropics.

Minnesota. Head,[35] in 1904, reported a case originating in Maple Lake, Itasca County, and in 1905 Aurand[36] a case in a millworker who had lived in Minneapolis many years and had never been South.

Montana. Spelman and Wherry,[37] in 1906, reported a case which was unquestionably infected in Montana.

Arkansas. One of West's[38] cases was probably infected in this state.

Indian Territory. West contribted one case from here.

Texas. Boggs has contributed to the literature two cases. Hektoen[39] has reported one, Dock,[40] in one report, one case, in a second,[41] twelve, Diamond[42] two cases, and West three.

The review of the literature on this subject shows, therefore, that the prevailing idea concerning the source of amœbic infection is hardly justifiable. Probably this erroneous belief has been responsible for failure to recognize the disease. A realization that this type of disease is endemic in New York led to a search for it, with a result that these three cases were found within a year. It is to be hoped that a realization of its existence elsewhere will give rise to the recognition of cases and to the establishment of proper prophylactic and therapeutic measures.

BLOOD CULTURES IN HUMAN GLANDERS.

By Burrill B. Crohn, M.D.,

FORMERLY PATHOLOGICAL INTERNE AT THE MOUNT SINAI HOSPITAL, NEW YORK.

The invasion of the blood current in cases of glanders in human subjects is of considerable scientific interest from three points of view: (1) By reason of the light which it throws on the pathological processes of the disease and the modes of dissemination of the infection; (2) on account of the valuable theoretical suggestions in

[31] St. Louis Medical Review, 1902, xlvi, 55.
[32] Ibid., 19.
[33] Ibid., 1903, xlviii, 343.
[34] Journal of the American Medical Association, 1894, xxii, 149.
[35] Northwest Lancet, 1904, xxiv, 249.
[36] Ibid., 1905, xxv, 321.
[37] Ibid., 1906, iv, 277.
[38] Trans. First Pan Amer. Med. Cong., 1893, i, 301.
[39] North. Amer. Pract., 1892, iv, 554.
[40] Medical Record, 1891, xl, 7.
[41] Quoted by Harris.
[42] Philadelphia Medical Journal, 1900, v, 817.

relation to infections in general, their mode of establishment, and means of propagation; and (3) by reason of the diagnostic aid given in cases of infection with indefinite or unrecognizable origin. The marked progress recently made in the study of blood invasions by bacteria has assisted materially toward an understanding of the sequence of events and rationale of diseases in which the circulatory medium is invaded by pathogenic organisms, either primarily or secondarily. Certainly, blood cultures in such diseases as typhoid fever, acute bacterial endocarditis, infective venous thrombosis, etc. have thrown new light upon the clinical course of the diseased processes, and have helped materially in advancing a more logical mode of studying and treating these cases.

Human glanders may safely be regarded as another of the bacterial diseases in which the blood current, sooner or later, is invaded by disease producing bacteria. It is still more probable that this fact plays not a secondary, but an important and essential part in the dissemination of the infection.

Soon after the first conception of the bacterial nature of certain diseases was established, and coincident with the discovery by Loeffler,[1] in 1882, of Bacillus mallei, glanders was regarded as an infection of pyemic nature. This conclusion was readily drawn by clinicians and was founded upon the simple observations that the disease began in a primary focus of infection, and that it pursued its course with the early establishment of foci of suppuration far distant from the orginal point of inoculation and apparently not transmitted by the lymphatics or by cutaneous auto-inoculation. As in these metastatic deposits, the original offending organism, as seen in the primary focus, was also readily demonstrable, the natural conclusion was drawn that the blood current was the medium of transmission. In support of this, the older pathologists in the era before the introduction of blood cultures, demonstrated the fact that in almost all the autopsies on cases of this nature, Bacillus mallei could be seen in smears of the blood after death (Koch,[2] Wassilief,[3] Kiemann,[4] Babes[5]).

The earliest attempts to recover the bacilli from the circulating medium were unreliable because imperfect methods were used. The first success was that of Frese, Ross, and Wilbert,[6] who, in 1891, succeeded in recovering the living organism from a patient suffering from the acute form of the disease. Since that date, and with the advent of improved methods, there have been sixteen cases in which the blood from the patient has been cultured (see Table). The results are fairly uniform in character, and the cases group themselves into two classes: (1) Acute glanders with positive or negative blood cultures, and (2) chronic glanders.

[1] Arbeit a. d. Kais. Ges., 1886, Band i, b. 141.
[2] Arch. klin. Chir., lxv, 37.
[3] Deut. med. Woch., 1883, p. 155.
[4] Wien. med. Woch., 1888, i, 515.
[5] Arch. de méd. exp., 1891, iii, 619.
[6] Medical News, 1891, p. 679.

Case No.	Author	Clinical Data			Cultural Data			Result
		Onset	Symptoms	Course	Day of culture	Symptoms at time of culture	Technique of culture	
I	Frese, Ross, and Wilbert: Med. News, 1891, p. 679	Pain in right shoulder. (Occupation, driver)	Rapid pyemia; pustules; nasal mucosa and respiratory mucous membrane involved	17 days	14th	Before general pyemic demonstration; pain in shoulder only symptom at time of culture	Blood taken in small glass cell, examined microscopically, and cultured	+
II	Duval, Gasne, and Guillemont: Arch. de méd. exp., viii, 361	Onychia on hand. (Occupation, hostler)	Temperature, 102.2° F.; pulse, 100; eruption on tenth day after admission; pustular eruption on eleventh day after admission	36 days	33d	Temperature, 100.2° F. to 104° F.; pustular eruption on previous day	Tubes of gelatin and bouillon inoculated; on fifth day one colony; 10 c.c. of blood (not all used)	+
III	Brault and Rouget: Gaz. hebd. de méd. et chir., 1896, p. 1212	Malaise for several days	High temperature; vomiting; lungs negative; abscesses on thirteenth day of disease; rales in chest; fever; chills; pustular rash on nineteenth day of disease	20 days	17th	No pustular rash until two days after culture; chills; fever; metastatic foci	?	+
IV	Coleman and Ewing: Jour. Med. Res., 1903, iv, 223	Bitten on hand by horse three weeks before; headache; malaise	Abscess of leg on eleventh day; pustular rash on twelfth day; pyemic foci (thirteenth day); autopsy, glanders and farcy	17 days	16th	One day before death, pyemia	Five c.c. of blood put into bouillon and kept on ice for two days; late flocculent growth	+
V	Denis: Lyon Méd., 1903, i, 664	Pain in right hypochondrium; fever; chills	Muscular abscesses; pyemia; pustular rash three days antemortem	27 days	24th	Pustular rash appeared same day as culture was taken	Bouillon flasks	+
VI	Gabrielides and Remlinger: Compt.-rend. hebd. de soc. de biol., 1902, p. 1147	High fever; typhoid state (Occupation, gardener)	High fever; pectoral abscess; typhoid state	26 days	23d	Complete pyemia	?	+
VII	Van Ingen: Med and Surg. Reports, Presbyterian Hosp., 1904, p. 320	Malaise for three weeks; delirium	Bronchopneumonia; swollen knee; patches in mouth; pyarthrosis; pustular eruption one day ante-mortem	32 days	23d 24th 30th	Broncho-pneumonia; pyemia; pustular rash on twenty-ninth day; positive blood culture on thirtieth day	?	— — +

VIII	Lewis and Duval: Jour. Med. Research, 1904–05, viii, 535	Pain in chest; chills; fever. (Occupation, hostler	Bronchopneumonia; general pyemia; pustular rash	12 days	10th	Complete pyemia; pustular rash	Agar plates; colonies on third day, 150 to 200 to every plate	+
IX	Pilcher: Annals of Surgery, March, 1907, p. 444	Bitten six weeks previous by glandered horse; pain in chest; cough; dyspnœa	Pneumonia; knee involvement on seventh day; general pyemia on ninth day; autopsy; broncho-pneumonia; pyemia; ulcer of larynx	13 days	10th	Pyemia; pneumonia	Agar plates; 48 hours, 35 colonies per plate; raised, white, opaque, viscid; flasks show flocculent sediment	+
X	Meyer and Crohn: Jour. Amer. Med. Assoc., May 16, 1908, p. 593	Patient lived over stable	General pyemia; rales heard in chest; deep muscular abscesses; pustular rash	11 days	7th 9th	Pyemia; pustular rash before cultures	See text (Technique)	+ +
XI	Unpublished case. (See text)	Sudden onset; fever	Deep muscular abscess over shoulder; pustular rash	13 days	13th	Deep prostration; one metastatic focus; pustular rash same day	See text (Technique)	+
XII	Gutowski: Arch. gen. med., 1903, ii, 2186	Pain in right side; pleurisy; malaise	Pleurisy; Bacillus mallei found in pleural fluid; superficial pustules; deep muscular abscesses	27 days	13th	First pustules and first abscess on eleventh day; delirium; profuse rash on thirteenth day	?	—
XIII	Potter: Proc. N. Y. Path. Soc., i, 120	Pleurisy; abdominal pain; fever	Pleurisy; first metastatic focus on fourteenth day of illness; autopsy, glanders and farcy of nasal mucosa, etc.	35 days	21st	(Day of admission)	?	—
XIV	Woodcock: Lancet, 1906, i, 288	Acute pyemia	Rapid acute pyemia beginning in lung; pustular rash	...	...	Day before death	Unsatisfactory bacteriological study	—
XV	Post: Boston Med. and Surg. Jour., 1905, cliii, 580	Pneumonia; small multiple abscesses	Multiple abscesses; no rash; pneumonia; case has lasted nine months; still living at time of publication of paper	9 mos.	...	?	?	—
XVI	Sieur: Arch. de méd. et phar. militaire, 1905, xlvi, 313	Inoculation of finger	Chronic pyemia	2½ yrs.	...	?	?	—

ACUTE GLANDERS. This series includes 14 cases, of which, 11 showed positive and 3 negative blood findings. All of these cases, those with positive and those with negative results, clinically follow rather closely one type of the disease. There is almost invariably a history of exposure to equine sources of infection; in 4 cases there was an actual point of inoculation on the hand. The onset is acute, usually with sharp pain in the lower thorax, rapid development of chills, fever, malaise, general prostration, etc. Initial symptoms referable to the lungs are found in over 80 per cent. of the cases. The picture of a general pyemia is gradually superimposed, or presents itself early in the course of the infection. Numerous subcutaneous and intramuscular and arthritic foci of suppuration appear. There is increasing prostration and rapidly supervening delirium, until the breaking out of a generalized pustular rash heralds the closely following fatal issue. The duration of the disease varies from eleven to thirty-six days. The picture is invariably one of a profound pyemia. Pulmonary involvement, either early and probably primarily, or later and secondary to a distal point of inoculation somewhere on the body, is invariably encountered. The localization of the infection, however, in the upper respiratory passages, so characteristic of glanders in the horse, is rarely seen in man. In the latter the picture of farcy, that is, accentuation of the localized point of inoculation with rapidly developing blood invasion and metastatic deposits, supervenes.

As has been noted, of the 14 cases of acute glanders with blood cultures, 11 were positive and 3 negative. A more detailed scrutiny of these findings reveals the following: Of the positive cases, the blood culture was obtained in almost all, when the disease was well advanced, that is, in from one to three days antemortem; in one case (Case X), it was taken four days before death. Considering that the total duration of the symptoms varied from eleven to thirty-six days, it is clear that the positive culture occurred late. In all but one case (Case I) there were already established numerous metastatic abscesses and active pulmonary phenomena; in 7 of the 11 the pustular rash had already made its appearance. In all the cases at the time of culture, there was profound prostration, high continuous fever, rapid pulse, and signs of intense generalized infection.

Of the three negatives cases, that reported by Woodcock (Case XIV) can be disregarded, as the subsequent bacteriology of the whole case was not satisfactorily nor conclusively worked out, nor was the organism accurately identified. Regarding the other cases with negative results, it is to be noted that they correspond in their clinical aspects fairly closely with the above series of cases with positive cultures. The only apparent difference is in the somewhat protracted course (twenty-seven and thirty-five days) and the less rapid appearance of the disseminated metastatic foci. In these two cases the attempt to isolate the organisms from the blood was made

relatively earlier in the course of the symptoms as compared with the previous series; in Case XII on the thirteenth day (total duration twenty-seven days), in Case XIII on the twenty-first day (total course thirty-five days). In neither case was the generalized pustular rash evident at the time of the culture.

The case reported by Van Ingen (Case VII) is of unusual interest for the reason that the blood cultures taken respectively on the twenty-third and twenty-fifth day of the disease were negative, though lung involvement and numerous metastatic abscesses were present. The culture taken on the thirteenth day (forty-eight hours antemortem), and the day just preceding the outbreak of the pustular rash, was positive.

Chronic Cases. Of two cases, both had negative blood cultures. That of Post (Case XV) had run a course of nine months with pulmonary and metastatic foci, but no pustular rash. The patient was still living at time of publication of his paper. That of Sieur (Case XVI) ran a course of two and one-half years, following a local infection of the finger, and presented the symptoms of chronic pyemia without pulmonary or cutaneous signs except toward the very end.

Technique and Technical Results. These have varied considerably in the past, and show numerous wide discrepancies according to the dates and degree of development of the bacteriological methods in vogue at the time. In general, the blood was drawn from the median or saphenous vein and inoculated immediately into either bouillon or agar or gelatin. (In Case IV the blood after being mixed with bouillon, was kept on ice two days before being incubated.) The results seemed good with any of the media used. Colonies appeared rather late (on the third to the fifth day) on the plates. No type colony has been described. In several of the cases the findings were complicated by the simultaneous growth in the culture of streptococci or Staphylococcus aureus, this being in all probability a secondary and late invasion of the blood from some of the numerous superficial and deep foci of suppuration.

It may be of some interest to recount the methods and results used in obtaining cultures in our own cases (Cases X and XI). In Case X (there were two blood cultures). In the first culture 15 c.c of blood was evenly distributed into a flask of plain and 2 per cent. glucose bouillon and into a plate each of agar, serum agar, glucose agar, and serum-glucose agar. Growth appeared at the end of forty-eight hours in both flasks and in all the plates; there seemed to be no decided advantage in any one medium for the cultivation of the bacilli. The appearance of the flasks was as follows: Plain bouillon, after forty-eight hours, slight cloudiness; after seventy-two hours, a heavy murky sediment, the supernatant fluid being unchanged in color. The 2 per cent. glucose bouillon flask showed at forty-eight hours a slight reddish hue, which increased in depth until, after seventy-two to ninety-six hours, the whole presented a deep Bur-

gundy-red color with a heavy sediment at the bottom. The average number of bacilli per cubic centimeter (as estimated from the plates) was 1.6 colonies.

The second blood culture on the same case was performed with identical methods, except that glycerin agar was added to the mentioned media, and the flasks were omitted. Growth again appeared relatively early, in forty-eight hours, the serum-agar plate showing the earliest and most profuse growth. Some blood that had been inoculated into ammonium oxalate[7] after incubation for twenty-two hours was plated out with agar, and showed after forty-eight hours from two to four times as many colonies as were found in the plate that was poured in agar at the bedside.

The colony, as appearing on glucose agar, with or without serum, may be described as a minute punctate (surface) or whetstone shape (deep) colony, of the size of a pin-point, or very slightly larger, brownish by direct and grayish by reflected light, and surrounded by a relatively large greenish area of discoloration in the medium about the colony. This discolored area, when first seen, is 2 to 5 mm. in diameter and increases in circumference rapidly. The morphology of the colony is noteworthy on account of the disproportion between the minute, almost invisible point of growth, and the large spreading greenish area of discoloration. When typical, it resembles very closely that of the typhoid-type colony described by Epstein[8] on glucose-agar plates. In the second culture in this case the average number of organisms per cubic centimeter of the blood was 16.8 (previous culture, 1.6), showing a rapid increase in the number of organisms in the circulating blood in the interval between the two cultures (two days).

The blood culture on the second of our two cases (Case XI) was made similarly to the above-described culture but taken on the operating table a few hours before death. The type of colony on the media was similar to the above; the time of appearance was somewhat earlier, the first colony being observed seven hours after inoculation. The average number of colonies was 300 per cubic centimeter.

From our experience (limited as it is) it would seem that the best results are obtained with the flasks and glucose-agar media; the ammonium oxalate method may apparently be used with safety.

REMARKS. The conclusions that may be drawn from the study of the cases of human glanders in which blood cultures have been taken are as follows: In the acute type, cultivations from the blood early in the course of the disease are likely to be negative; those taken at a time later in the development of the symptoms, particularly at or after the time of the appearance of the pustular rash, are invariably positive. Chronic cases have thus far given negative results.

[7] Epstein, AMER. JOUR. MED. SCI., September, 1907.

[8] Ibid., August, 1908.

These conclusions are singularly in contrast with the results obtained by investigators of equine glanders. Thus, MacFadyean,[9] an eminent authority on the subject, says: "The glanders bacillus is a tissue parasite, and in all cases of glanders in the horse it is almost entirely confined to the lesions and the discharges from them. It is, no doubt, transported by the blood as well as the lymph channels, but the disease in the horse is never septicemic. Even in the acute cases the bacilli are so sparingly present in the blood that their discovery by microscopic and cultural methods is nearly always impossible, and even considerable quantities of blood fail to transmit the disease by inoculation." The explanation of this difference lies in the fact that the disease in the horse, rarely, if ever, runs so acute a course as it does in man, manifesting itself in the former mainly as a subacute or chronic affection of the respiratory apparatus or as a localized farcy of the skin and related lymph organs. The corresponding type in man, that of the so-called chronic glanders, also gives negative blood cultures. The usual picture of the infection in the human is, however, that of a rapidly developing and grave pyemia with a less considerable and more slowly developing involvement of the related lymphatic channels, but with the rapid evolution of metastatic muscle, bone, and joint foci which have, in all probability, been transmitted through the circulation. It is quite probable that bacteria are present in the blood in human cases early in the disease; in fact, as early as the first metastatic deposit has made its appearance, but they are in such few numbers that with our present methods we are incapable of demonstrating them. It is in the later stages of the disease, when the bacteria become much more numerous in the circulation (as evidenced by the outbreak of a general pustular rash, the pustules containing bacilli), that we are successful in cultivating them. In the chronic cases, those which present the picture of generalized glanders of the respiratory tract, the blood is probably sterile. In those cases with the establishment of farcy buds it would perhaps be possible to cultivate the organisms from the circulation, if the attempt were made just at the time of the new outbreak or at the time of a new deposit of the infection. Between the eruptions of the peripheral foci, attempts at isolating the specific organism from the blood would be, in the greatest probability, unsuccessful.

As to the nature of the bacillemia, the question of chief importance is: "In what manner, do the bacteria enter the blood and circulate therein?" Is it an overflow from the lymphopoietic system? Is it a condition of thrombus formation with multiple minute emboli? Is it a true bacillemia? These are the three views which would seem most tenable. In discussing the bacillemia of typhoid fever, Schottmueller[10] and, later, Coleman and Buxton[11] have suggested that the

[9] Jour. Comp. Path. and Ther., 1904, p 295.
[10] Münch. med. Woch., 1902, No. 38.
[11] Amer. Jour. Med. Sci., June, 1907, p. 869.

bacillemia is secondary to the infection of the lymphoid apparatus of the gastro-intestinal tract, and is an overflow from the lymphopoietic system. Whether the infection of the blood stream in glanders may be the result of an overflow from the lymph system, which is invariably involved with the local lesion, is seriously to be considered. Here we must understand that the lymph gland has no longer become capable of maintaining its function as a filter for the regional lymphatics, and the organisms passing this barrier are swept into the general blood current and so enabled to establish the metastatic foci. If we accept the hypothesis of the possibility of a primary infection of the lungs and respiratory tract by inhalation, we must suppose the bronchial glands to be the source of overflow in most of our cases.

On the other hand, the recent work of Duval,[12] on "Vascular Lesions in Experimental Glanders," has demonstrated that there is a marked reaction of the vessel wall to the specific toxin, and that thrombosis of the venous radicals in the neighborhood of the point of inoculation does occur. In the light of this fact, a priori, it may be supposed that the metastatic foci and free bacteria have a common origin in these thrombi and that the distal deposits result from minute thrombotic emboli. In opposition it must be maintained, that it would be an unusual and rare variety of embolus which chooses the deeper sets of muscles and joints as its site of preference and omits those organs usually the seat of infarcts (spleen, kidney, lungs). Emboli arising from foci of inoculation on the extremities or upper respiratory mucous membrane (nose, pharynx, larynx) should cause infarction in the pulmonary artery system and the lungs, and emboli arising from pathological processes in the lungs would be expected to cause the more usual infarction of the larger abdominal organs.

The third view, that it is a true bacteriemia, should also be taken into consideration. According to this view, we should understand the disease as one in which the bacteria, by injuring with its toxins and so depressing the vitality of the vessel wall, escape into the blood current and establish metastatic foci in those places specific to the glanders bacillus. Probably early in the disease, with a beginning local lesion and a mild degeneration of the vessel wall, there is only a scant, if any, escape of bacteria into the circulating medium, those few cases in literature in which the culture was taken early, neglecting to show the organisms. Further, with the advancement of the lesions and probably inevitable involvement of the respiratory tract there is a greatly increased escape of bacteria through pervious vessel walls, and possibly an active multiplication by reproduction of those organisms free in the current.

On account of the paucity of records and the few cases in the literature, no absolute deduction can be drawn as to the origin and

[12] Jour. Exp. Med., 1907, ix, 241.

course of the bacteriemia. With our improving methods for early recognition of the condition, and with more frequent and earlier cultures, the future will probably contribute much toward the solution of the problems connected with this rare but interesting disease.

The following is the history of the case referred to as Case XI:

The patient, a female, Russian, aged forty-three years, was admitted to the Mt. Sinai Hospital (service of Dr. Gerster), September 22, 1908.

The family history was negative and the previous history uneventful. The patient was married at twenty-seven years of age; had seven children; no miscarriage; she had fever lasting three or four days after the last labor. The present illness began suddenly nine days before admission with chills, fever, and vomiting. She had headache and profuse night sweats accompanied by chills. She vomited clear fluid; not bloody. Synchronously with the onset of febrile phenomena she developed severe pain in the left shoulder with loss of function of the upper extremity on this side. The pain was localized to the shoulder-joint and scapular region, was very intense, boring in character, and severe enough to prevent sleep. Since the onset the fever had slightly subsided, though the local focus continued very tender and painful. She had no abdominal symptoms, and no jaundice.

Upon physical examination on admission the general condition was fair; she was poorly nourished. The glands were not enlarged. The skin was negative, except over the scapular focus, where the swelling was surrounded for a small area by a few raised pustules, each pustule being circumscribed by a small red ring of inflammation. Active motion in the left shoulder was impossible; passive motion unimpaired, but painful. Over the left scapula was a smooth, rounded, firm swelling; no fluctuation was obtained. The skin over the swelling was reddened and elevated. There were cup marks over the swelling and signs of an aspiration wound. The lungs and heart were negative. The pulse was rapid, of good volume, diminished tension, 90 to 96 in rate. The abdomen was negative. There was a profuse, blood-stained vaginal discharge.

September 24. Aspiration of the tumor over scapula by Dr. A. A. Berg. Only a little blood was obtained, although the needle touched bare bone. Blood count: White blood cells, 22,000; polynuclears, 90 per cent.; lymphocytes, 9 per cent.; eosinophiles, 1 per cent. The pulse rate varied between 118 and 128. The temperature was remittent in character, reaching its highest point at about four in the afternoon (104.6° F.), and its lowest point at about four in the morning, when it was 100.2° F. Respirations varied between 24 and 28; regular.

September 25. Blood count: White blood cells, 9000; polynuclears, 91 per cent.; large lymphocytes, 5 per cent.; small lymphocytes, 4 per cent. The pustular eruption has spread from the

scapular region so that it now is more general in character and involves the neck, back of the extremities, and the entire dorsal surface of the trunk.

Operation, September 26: Incision and drainage of acute suppurative myositis. Dr. Berg. A four-inch incision over infraspinous fossa down to bone. Intraspinous muscle is found to be the seat of innumerable miliary abscesses varying in size from pinhead to the size of a grape seed. Multiple incisions into the muscle and excision of a piece for examination. Wound packed with gauze; wet dressing.

The patient's condition was rather poor on leaving the operating room. Soon after, respirations became more regular and strong, the pulse good at the temples, but absent at the wrist. Shortly the patient had a severe chill. The condition became quite poor. She re-acted poorly to vigorous stimulation. Murphy infusions proved ineffectual. She vomited several ounces of brownish fluid. Stomach tube was passed and stomach washed; six ounces of bloody fluid was withdrawn, but the patient immediately collapsed and ceased to breathe after five minutes.

Urine, September 23: Amber; turbid; acid; 1020; very heavy cloud of albumin; no sugar; loaded with hyaline and finely granular casts. September 26: Amber; acid; 1022; heavy trace albumin; many granular and hyaline casts; few epithelial and pus cells.

This patient was in the hospital four days. No evidence of exposure to glanders in any form was obtained. The local focus in the left shoulder was apparently present from the onset. The infection was severe, the course rapid. There was general prostration and the toxemic reaction was intense. Even as early as nine days after the onset the beginnings of the pustular rash were seen. It is noteworthy that in this case there was no other metastatic focus established as far as could be ascertained. The blood culture was taken on the operating table. It will be noted again that the blood culture was taken late in the course of the disease after the appearance and spread of the pustular rash. There was no involvement, as far as could be discovered, of the lungs or any part of the respiratory tract. Permission for an autopsy was refused.

Bacteriological Data. Smears from pus removed at operation showed the morphology of Bacillus mallei. Emulsion of the same material inoculated intra-abdominally into male guinea-pigs caused a characteristic reaction. The organism was further identified by staining methods and cultural means. For notes on the blood findings see under "Technique."

I am indebted to Dr. E. Libman for many valuable suggestions and for his kind guidance throughout the course of the investigation.

A SIMPLE METHOD OF ESTIMATING THE COMMON VARIATIONS AND DEFORMITIES OF THE FOOT.[1]

By Henry O. Feiss, M.D.,
ASSISTANT SURGEON TO THE OUT-PATIENT DEPARTMENT OF THE LAKESIDE HOSPITAL, CLEVELAND, OHIO.

Present Methods of Studying the Foot. There is no part of the human body so subject to deformities as the foot, yet there is no part of the body of which the estimation of deformities is so inaccurate and vague. The most common deformity that we see is flat foot, yet the diagnosis of flat foot, so far as its physical signs go, rests on the most ambiguous basis, and in attempting to teach students what we mean by the term, we seldom are able to present a convincing and definite picture. The only method which pretends to any accuracy at all is that of taking a print of the bottom of the foot. The deductions thus made are dependent on the extent with which the sole of the foot touches the underlying surface. Then the shape of the print is supposed to be an index of the lowering of the arch. I see little value in this method. In the first place, the print of the foot only represents the imprint of the skin of the sole. If a foot happens to be fleshy or swollen, it naturally makes a broader impression, and thus seems to connote a lower arch than when a foot is thin and not swollen. Again, a foot which may be very flat may make an almost perfect print, so called, if its arch has not been lowered quite enough to be represented properly in the print. But even if one admits that this method does aid in diagnosis in extreme types of the disorder, it adds nothing in estimating the amount of lowering of the arch in those types which are intermediate. Therefore, in the last few years I have sought for a more definite basis for comprehending certain deviations, with special reference to the estimation of the height of the arch; the aim of this paper is to suggest such a method.

The Estimation of the Deformity in General. In examining the malformations of the body there is no process more valuable than that of studying the relationship of bony landmarks to one another. In fractures of the wrist and of the ankle the relationships of the ends of the long bones are of extreme importance. A splendid example of valuable relationships is Nélaton's line. I may further call attention to such important methods as Bryant's triangle and Meyer's line. These simple geometric methods can hardly be omitted for a clear and proper elucidation of the deformity.

The Selection of Bony Landmarks of the Foot. In the case of the foot no such method has ever been advanced, so far as I know,

[1] This paper represents the substance of a monograph which the author is preparing, in which the work will be presented in greater detail.

and with this in mind I have selected certain landmarks for study. The ones chosen are the lower posterior corner of the internal malleolus, the tubercle of the scaphoid bone, and the lower tubercle on the head of the first metatarsal. The reason for selecting these particular landmarks will be more evident as we proceed. Before advancing, let me briefly examine these landmarks, as it is absolutely essential that the observer know definitely just what part of the skeleton his finger touches in the examination. Fig. 1 is a wash drawing of a disarticulated skeleton which brings into exaggerated relief the landmarks in question.

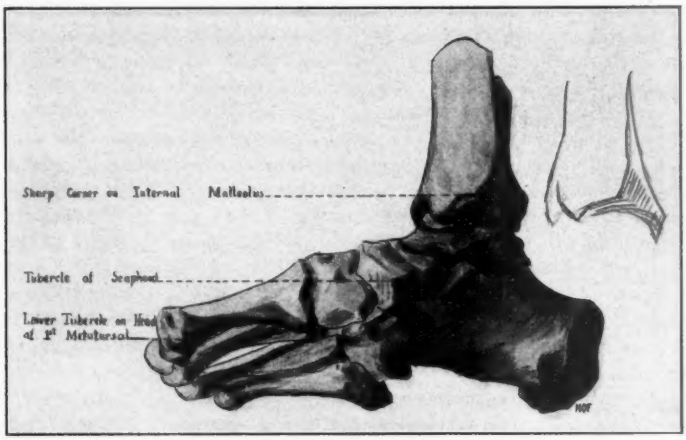

FIG. 1.—The position of the bony landmarks under consideration.

The Lower Tubercle of the Head of the First Metatarsal Bone. In examining the head of the first metatarsal bone in the disarticulated skeleton, we find on its inner aspect two ridges separated by a groove. Each of these ridges ends posteriorly in a prominent tubercle. The upper tubercle does not concern us; the lower one is sharp, and is the one which applies to the present series of observations.

The tubercle of the scaphoid bone is best seen by looking at the scaphoid bone from above. It will then be apparent that the whole internal surface of this bone forms a ridge. The apex of this ridge is the so-called tubercle, being, in fact, the most prominent bone palpable. The tendon of the tibialis posticus ends close to this tubercle.

The Posterior Inferior Corner of the Internal Malleolus. As seen in the sketch, and as will be apparent on examining the skeleton, the internal suface of the tibia is bounded at its lower end (internal malleolus) by three edges—an anterior, an inferior, and a posterior edge. Where the posterior edge strikes the inferior edge a sharp

corner can always be felt. If we run our finger over the part in question we simply determine the most inferior posterior corner of the internal malleolus as thus evidenced.

CHANGES IN THE SKELETON OF THE FOOT WITH WEIGHT-BEARING. The investigation of the foot differs from that of the other parts of the body in one respect—that the relationship of the bones becomes altered with weight-bearing. Their alteration is necessarily the first study which most concerns us. As similar studies have been made by other observers, I shall confine myself to the change in the relationship of the landmarks in question.

According to the literature,[2] the view seems pretty well established that when weight is borne the scaphoid travels downward and inward. The downward displacement is, for our purpose, the important thing. In the study of six living feet which I radiographed, with and without weight-bearing, I marked the posterior inferior corners of the tibia, the head of the first metatarsal, and the scaphoid. (The external landmarks selected cannot be accurately identified in the Röntgen pictures.) I then drew a connecting line between the point marked on the internal malleolus and that on the head of the first metatarsal. Figs. 2 and 3 illustrate one of these cases and show that in weight-bearing the scaphoid becomes distinctly depressed with respect to the connecting line. However, as these points on the Röntgen pictures do not exactly correspond to the external landmarks which I selected (earlier described), I marked these landmarks with and without weight-bearing and photographed the feet (Figs. 4 and 5). These pictures point to a similar but more practical conclusion, that in weight-bearing the scaphoid becomes depressed with respect to the line connecting the lower posterior corner of the internal malleolus with the lower tubercle on the head of the first metatarsal.

It is, therefore, evident that in selecting the scaphoid bone, I have taken a part of the foot very susceptible to change in weight-bearing, and on a basis of the above observations and others,[3] not described in this report, I may state that the weight-bearing foot differs from the non-weight-bearing foot according to a definite rule. This rule is that in weight-bearing the scaphoid descends, as measured from the base line as established above.

THE STUDY OF THE POSITION OF THE SCAPHOID TUBERCLE IN ONE HUNDRED YOUNG MALE ADULTS. After having studied the anatomical data above described, I examined a number of apparently

[2] G. H. V. Meyer, Statik und Mechanik des menschlichen Knockengerustes, 1873, and Statik und Mechanik des menschlichen Fusses, 1886; John Dane, Trans. Amer, Orth. Assoc., 1897; Golobiewski, Ztschr. f. orth. Chir., 1894; Lovett and Cotton, Trans. Amer. Orth. Assoc., vol. xi; Bradford and Lovett, Text-book of Orth. Surg., second and third Editions; Whitman, Orthopedic Surgery; and Quain's Anatomy.

[3] In the monograph another method of measuring the excursion of the scaphoid in weight-bearing is described. This consists in using the angle which it forms with the vertical dropped from the internal malleolus The results so obtained confirm the main observation.

healthy individuals with reference to these points. The individuals chosen for the observation were young male adults from the evening

FIG. 2.—Röntgen picture of a foot without weight-bearing: *c*, marks the posterior inferior corner of the head of the first metatarsal; *a*, the posterior inferior corner of the internal malleolus, and *b*, the posterior inferior corner of the scaphoid. A vertical line is erected from *b* to *a-c*.

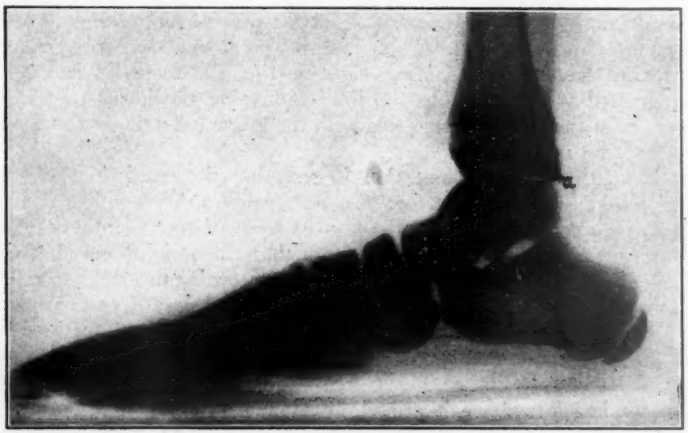

FIG. 3.—Röntgen picture of the same foot as in Fig. 2 with weight-bearing. The same points are marked. Note the increased distance of *b* from the connecting line.

gymnastic class of the Cleveland Young Men's Christian Association. The men were taken as they came along, and no questions were asked. It may be assumed that such a gymnasium would furnish

FIG. 4.—Photograph of the same foot as in Figs. 2 and 3, without weight-bearing. The posterior inferior corner of the internal malleolus, *a*, is connected with the lower tubercle on the head of the first metatarsal, *b*, by a line; *c*, represents the scaphoid tubercle.

FIG. 5.—Photograph of same foot as in Figs. 2, 3, and 4, with weight-bearing. Note the increased distance of the scaphoid tubercle from the connecting line.

a representative group of healthy young men who conduct the ordinary affairs of life.

In order to make proper observations, a standard method of estimation had to be applied. A wooden box was constructed, consisting of three sides—a base, a posterior side, and a lateral side. Its dimensions were 12 x 12 x 5 inches. The box was placed on the seat of an ordinary chair. The right foot was used. The subject stood on the floor with the left foot straight and the right knee bent. Then the right foot was placed in the box, the calf just touching the upper edge of the box, and the heel resting in the corner (Fig. 6).

FIG. 6.—Position of the foot in the box.

The weight of the body was therefore chiefly transmitted through the left leg. In this way the foot could be examined in a definite and uniform position without weight-bearing. The points marked were those landmarks earlier discussed—the posterior inferior corner of the internal malleolus, the tubercle of the scaphoid, and the lower tubercle of the first metatarsal. The distance of the scaphoid tubercle from the line connecting the internal malleolus with the lower tubercle of the head of the first metatarsal was measured as follows: A thin transparent celluloid rule provided with a scale (Fig. 7) was applied to the foot so that its upper edge coincided with the dots on the head of the first metatarsal and on the corner of the internal malleolus. Then the distance of the tubercle of the

scaphoid from the edge could be read through the transparent rule by means of the scale (the corners of this rule were cut off so that they would not interfere with the sides of the box).

SUMMARY OF IMPORTANT RESULTS. The average age of the one hundred young men was found to be 21.6 years, the youngest being seventeen and the oldest thirty-four years. The average distance

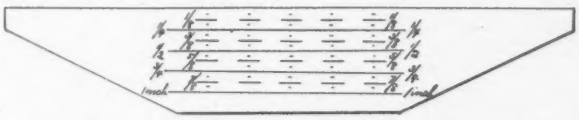

FIG. 7.—The transparent celluloid rule used for determining the distance of the scaphoid tubercle from the connecting line (reduced one-half).

of the tubercle of the scaphoid from the line connecting the internal malleolus with the lower tubercle of the head of the first metatarsal was found to be 0.49 inch.

These results, though interesting, are of less value than the results obtained by arranging the cases in a series graded according to the lowering of the scaphoid tubercle. In the following table the cases were grouped in this fashion.[4]

Distance of the scaphoid tubercle from the line connecting the internal malleolus with head of first metatarsal.	Number of cases.	Distance from scaphoid tubercle to internal malleolus.	Horizontal distance of the tubercle from vertical line dropped from internal malleolus.	Sine of angle formed by line connecting the scaphoid with internal malleolus and the vertical line.	Angle.	
					Deg.	Min.
Inch.						
0	2	2.03125	1.71875	0.8461	57	47
1/16	2	1.875	1.53125	0.8166	54	45
1/8	5	2.0875	1.7	0.1843	54	31
3/16	0					
1/4	11	2.1136	1.6843	0.7956	52	43
5/16	3	2.0625	1.6458	0.7979	52	56
3/8	15	2.1666	1.6833	0.7769	50	58
7/16	2	2.0625	1.5	0.7272	46	39
1/2	19	2.2039	1.6052	0.7283	46	45
9/16	3	2.1666	1.5625	0.7212	46	9
5/8	18	2.125	1.5277	0.7189	45	58
11/16	1	2.0	1.4375	0.7187	45	57
3/4	15	2.3	1.5	0.6521	40	42
13/16	0					
7/8	1	2.375	1.5	0.6315	39	9
15/16	0					
1	3	2.5	1.666	0.6666	41	48
Average	..	2.15	1.59	0.7390	47	40

[4] Only the first two columns apply to the present text, but the other columns may be of interest.

The table shows that there are only nine cases in which the tubercle is less than one-fourth inch below the line, and only four cases in which it is more than three-fourths of an inch below the line. There fore, the great majority of cases show the tubercle depressed from one-fourth of an inch to three-fourths of an inch, which in itself is a large variation (one-half inch).

Conclusions from the Table. In studying the table we must remember that when there is a large variation within normal limits, conclusions based on only one hundred cases are of slight value; but the object of the observations was to determine the nature of the variation rather than its average extent. The important point is that the height of the arch as measured by its index, the tubercle of the scaphoid, does show this marked variation. This variation in a series of non-weight-bearing feet suggests the rule which applies to the individual foot with weight-bearing. In other words, the physiological variation (so far as the scaphoid tubercle is concerned) during weight-bearing function of the individual foot, is a prototype of the anatomical variation of a group without weight-bearing. This conclusion is one which has always been understood by the anatomists. It is simply a perhaps novel method of checking off the older observations; but the main question which is still open is, What is the normal foot? What kind of a foot are we to use on which to base the estimation of a deformity, and in what should a diagnosis of a pathological foot consist? These and allied questions require careful consideration and to this I shall now proceed.

The Average Foot. It was found that in one hundred cases the average depression of the tubercle of the scaphoid from the connecting line was about one-half an inch. As earlier stated, one hundred cases are too few on which to base a fair average. But even if thousands of cases had been used it would be fallacious to reason that all feet in which the scaphoid tubercle is higher than the average are normal, and all feet in which the tubercle is lower are abnormal, because the average is obtained just as much from the figures below as from the figures above. Moreover, the chief point shown in the hundred cases is that there is marked variation in the height of the scaphoid in apparently healthy feet. Consequently, as the average is based on that variation, it has no significance further than what the term indicates, representing simply an average of normal variation. Such an average, therefore, cannot serve as a basis from which to estimate deformity.

The Normal Foot. The problem is not what is the average, but what is the extent of variation which we have a right to expect in the normal physiological foot? In attempting to answer this question we deal with a problem in classification, the same problem which necessarily presents itself in the study of any series of graded objects. Given a series of objects animate or inanimate, which differ from one another only to a slight extent, it is impossible to

state just where we should draw the line dividing one group from another. If we take one hundred shades of color, say yellow, and arrange these shades in a graded series from a light yellow to a brown, the differences between the individual shades which are placed next to each other are so slight as not to be detectable. Yet if we compare the beginning with the end of the series, the difference is very striking. The same holds good in a given structure in the human body in which the individuals are arranged in a series according to the variations in that particular structure. If we take one hundred feet and arrange them in a series with reference to a given point, such as the height of the scaphoid tubercle, and attempt to base a classification on that variation, it is impossible to state where the normal variation ends and where a deformity begins, although the two extremes of the series are strikingly different. This, then, is our problem; the difficulty in dividing the series is apparent; there is no natural dividing line in this arrangement which can be used to define the limits of normality. I did, to be sure, find that the great majority varied between one-fourth and three-fourths of an inch, yet I can lay no stress on these figures, because it is evident that if more than one hundred subjects had been used, the gaps between the figures would have been filled up. Nevertheless, as will be seen, such an arrangement in a graded series is of considerable importance, even if the dividing line between the normal and the pathological does not exist, because this method will help to organize our knowledge on the subject.

THE "TYPE" FOOT. It has been shown that the average foot is not a fair basis from which to estimate a deformity, and that it is not possible to make a sharp distinction in the series for purposes of classification. What, then, is a sensible method for systematizing our views? The only course which remains is to make an artificial distinction between the normal and the abnormal, but an artificial distinction which is not only practical, but which has a reasonable theoretical basis. If we study the table of the one hundred cases, we note the two extremes of the series, namely, the one extreme, in which the tubercle is not depressed at all, and the other extreme, in which the tubercle is depressed one inch. There can be no two opinions as to which extreme approximates more closely the physiological foot. There can be no doubt that the foot in which the tubercle is near the connecting line is a better foot than the foot in which the tubercle is one inch below the line. The reasons for this are twofold.

In the first place, as has been shown, the ordinary foot without weight-bearing has its tubercle higher than the same foot with weight-bearing. Now, we certainly know that the foot without weight-bearing has a greater strength and greater efficiency than the foot with weight-bearing, because the power of the former is not yet expended. On the other hand, the foot with weight-bearing is in a static position

in which part of its power is already used; consequently, a weaker foot for further purposes than the foot which has not expended its power. It seems, therefore, that other things being equal, the higher the scaphoid tubercle is, the greater the efficiency of the foot. Consequently, if we see a large number of feet all under the same static conditions, that is, all without weight-bearing, and find this variation of elevation of the scaphoid tubercle, it must mean that those feet in the group with the highest scaphoid (of which the prototype in the individual is the non-weight-bearing foot) must approximate the highest functional extreme.

In the second place, it is apparent from what has come before, and also from a number of healthy feet in which I studied the functional power (besides those used in the present investigation), that all those feet in which the tubercles were high with respect to the connecting line presented high arches and high insteps. It is, I admit, very seldom that the tubercle falls exactly on the line, but quite a few fall very close to it, either just above or below. All feet which showed this property I have found to be extremely well formed. In addition to the elevated arch and high instep, the proportions were extremely attractive and corresponded to what an art critic would deem a beautiful foot. Why is it that the artist considers such a foot beautiful? Simply because, as part of the body, it seems well adapted to carry on its functions. From his point of view, it is a more graceful thing than when it is not so well adapted. It is, therefore, plain that the commonly acknowledged beautiful foot is not acknowledged so because some men happened by chance to call the thing beautiful, but simply because it is the natural kind of foot which seems best fitted for its purpose. In short, both from the point of view of the physiologist and from the point of view of the artist, the foot with the high arch expresses strength and adaptability to function. Therefore, such a foot connoting strength and suggesting beauty, coming naturally at the extreme of a carefully selected series, must have some significance. Why not select this as an arbitrary type, remembering, of course, the method by which the type was chosen? If we do this, and bear in mind that it is only an artificial selection we have a fair starting point on which to base our estimation of deformity. Even if it is asserted that such a method is not completely scientific, it is at least reasonable, certainly more reasonable than having no method at all. A fairly good typical foot is illustrated in Fig. 6.

THE ESTIMATION OF THE DEFORMITY OF THE FOOT. Let me, therefore, suggest the following rule for estimating the deformity of the foot: Other things being equal, the foot is deformed in direct ratio to the deviation of the scaphoid tubercle from the line connecting the lower tubercle on the head of the first metatarsal bone with the lower posterior corner on the internal malleolus. Now, I am careful to state, "other things being equal," because I believe

that no estimation is of the slightest value without considering these other things. In any problem of this sort we have no right to take one physical sign by itself and make our estimation on that sign alone; we must always consider that point in relation to other clinical evidence. In flat foot, for example, we may have a number of considerations before us—the abduction of the foot, the pronation of the ankle, the prominence of the internal contour, etc. All these things may be correlative to the lowering of the scaphoid tubercle; if they are, we are probably dealing with a pathological foot, but if they are not, we must consider the relative value of each piece of evidence, depending in each case upon the individual circumstances of that case. I believe that the lowering of the arch is an important point in considering the deformity and that the best index for estimating the lowering of the arch is the scaphoid tubercle, but that is all the further I am willing to go. A lowered arch does not necessarily mean a deformity, it simply means a variation; but a badly lowered arch means deformity, and what is bad we must determine in the individual case. As will be shown (Case V), a foot may be very abnormal even if the tubercle is above the connecting line. The other physical signs in such a case are more pathognomonic than the height of the tubercle.

The Consideration of the Size of the Foot. With regard to the application of the rule to individuals of different size and age, I believe that its language is sufficiently broad to cover all ordinary cases, provided that one uses reasonably good judgment in the individual. Of course, an equal lowering of the scaphoid tubercle in two feet, one large and the other small, would necessarily be of greater significance in the latter. Thus a depression of the tubercle of three-fourths of an inch in a six-year-old child is of greater consequence than an equal depression in an adult.

The Definition of Flat Foot. It has been shown that the variation within normal limits in a group of feet is similar to the normal change in the individual foot with weight-bearing. From this it was naturally inferred that the more depressed a non-weight-bearing foot is, the less it is able to change with weight-bearing, and consequently that such a foot has lost part of its latent strength. Reasoning from this, I showed that a sensible classification could be made if we regarded such a foot as typical of highest function and beauty which most closely resembled the healthy non-weight-bearing foot, namely, one in which the tubercle is close to the connecting line, providing no other pathological sign co-exists. Flat foot, therefore, may be defined as a position of the bones, resembling that of physiological weight-bearing, which does not disappear when weight is removed, the amount of flattening being in the ratio of the lowering of the scaphoid tubercle.

From the above, it is clear that if we are to take such a foot as typical of beauty and function as has been described, then we can

consider few feet well formed, and that we must regard the great majority of human feet as flat. This is true; but if we regard them in this manner we must speak of the great majority as physiologically flat, or, better, as anthropologically flat. They are only flat as based on the estimation from a type, the type being based on the consideration of the highest function and beauty, and consequently of relatively rare occurrence. It therefore becomes clear that if only a few feet are best adapted to carry on their function from this point of view, and if the great majority of feet are more or less deficient as compared with the high standard that has been set, the diagnosis of flat foot can have little practical weight unless it connotes bad function and pain. If we consider an individual foot, and note that it is moderately flat, it does not mean that the foot is unable to carry on its function; it simply means that it might have been better if it were built more according to the type. If, however, there are signs of disability accompanying the signs of depression of the arch, such as pain and muscular spasm, then our diagnosis is established. We have a similar problem in the consideration of the physical signs of other parts of the body, for example in the consideration of hypertrophy of the heart. Hypertrophy of the heart may occur in apparently healthy individuals, and may not in itself cause symptoms; but once urge that heart to carry on functions beyond the power of compensation, and it loses its resistance to future dilatation much more readily than the heart which has not previously been hypertrophied.

From what has gone before, it is evident that the use of the bony landmarks of the foot as an aid to diagnosis must be made with the greatest caution. When one considers the extreme variation in the normal, when one considers the artificial manner of selecting one kind of foot as typical, one feels some hesitation in advocating the measure as an aid to diagnosis at all. Nevertheless, if the observer is careful, he may in many cases derive some value from such a method. But if he uses it, he can never lose sight of the value of the other points in diagnosis; he simply has one more physical sign to use as evidence. If he goes farther than this and bases his whole diagnosis on that one sign, he is likely to fall into grave error.

Clinical Application of this Method. If one follows such a suggestion as has been made, that of estimating the variation and deformity of a foot from an artificial type, it is necessary that the estimation should rest upon a standard method of examining the patient. Such a method has already been shown, namely, that of using a box of given dimensions, placing it at a given height, and using such a transparent ruler as earlier described. The only measure necessary is that of the distance of the tubercle to the connecting line. Simple as such apparatus is, even these things may be omitted for ordinary work. A method that I suggest is to let the patient stand on his one foot and to have him place the other foot

on an ordinary chair with the inner side of the foot facing the examiner. The weight of the body must be transmitted through the leg which is resting on the floor. Then the landmarks may be marked with a skin pencil (a good skin pencil is a grease paint pencil such as women use for painting their eyebrows), and then a simple ruler or piece of string may be applied to the points marked on the internal malleolus and the head of the first metatarsal. From this the distance of the tubercle can easily be measured. This is all the accuracy that is necessary, and requires no further apparatus. If such a box is used as has been described, it may be used both for the left and for the right foot simply by letting the patient stand on his other foot and by inverting the box. This being the method, the simple rule is: Other things being equal, the foot is deformed according to the deviation of the scaphoid tubercle from the line connecting the lower posterior corner of the internal malleolus with the lower tubercle on the head of the first metatarsal. In order to illustrate the practical applicability thereof, let me cite a few cases:

I. Feet Showing Asymmetry with Reference to the Elevation of the Scaphoid Tubercle. (*a*) *The Tubercle on the Unhealthy Side Lower than on the Healthy Side.*

Case I.—U. B., aged twenty-two years, by occupation a barber, was first seen at my office October 16, 1908. Three months ago the patient began to have trouble in his right foot after previous good health, this consisting in disability, pain, and swelling, and had grown steadily worse. There has been no history of injury. The left foot has not bothered him and has not become swollen. Examination showed the patient limping on his right foot, which was in a position of abduction. There was swelling in the region of the internal malleolus. The skin was somewhat clammy to the touch, and the region of the swelling was bluish white. There was marked stiffness on inversion. Examination of the relation of the tubercle of the scaphoid to the line connecting the internal malleolus with the head of the first metatarsal showed it to be seven-eighths of an inch below. The tubercle of the left foot was found to be five-eighths of an inch below the connecting line. There was no swelling and no signs of spasm in the foot.

It will, therefore, be seen that the lowering of the tubercle in this case, as compared with that on the other side, is correlative to other signs, but the amount of lowering is of considerable help, showing the anatomical difference between the diseased and the other foot. The diagnosis was acute flat foot (perhaps infectious) and irritated by occupation.

Case II.—E. B., business man, aged twenty-four years, was first seen by me September 21, 1908. Several weeks ago he strained his feet playing tennis; before this time he had had little or no trouble. Now there was pain in the right foot, especially on use, and chiefly referred to the arch and the ball of the foot Examination showed

the feet well developed, without spasm, swelling, or calluses. There was no redness and no tenderness. In the right foot, however, the tubercle was found to be seven-eighths of an inch below the connecting line, and in the left foot five-eighths of an inch below. The inner contour of the right foot bulged somewhat more than that of the left.

It is then apparent that if the measurement was accurately made, a slight deformity was present as compared with the other foot, and it is plain that the plantar ligaments must have been weakened and stretched.

FIG. 8.—Case III. Osteosarcoma. Note the complete obliteration of the arch out of proportion to the amount of lowering of the scaphoid tubercle.

CASE III.—F. F., aged thirty-nine years, a laborer, was first seen at the Lakeside Dispensary August 10, 1908. One year ago the right foot began to swell and the patient noticed his arch lowering. The swelling became worse and the arch grew flat. He had some pain and limped. He was treated for flat foot. Examination of the foot (Fig. 8) showed marked swelling, including the whole tarsus from the ankle to the metatarsal region. The foot seemed completely flat. The bony landmarks could not be distinctly felt. What seemed to be the tubercle of the scaphoid was one-half an inch below the connecting line; that on the other foot was about one-fourth of an inch below the connecting line.

Here we had an apparent incongruity of facts. There was bulging

of contour, marked flattening of the arch, and marked swelling, whereas the tubercle, which, if it were a flat foot, should have been markedly depressed, was only one-half an inch below the line. A Röntgen picture was taken and showed plainly that a mass of bone and new tissue had formed and included the whole tarsal region. The diagnosis of osteosarcoma was made. The seemingly contradictory evidence was the reason for taking the Röntgen picture, because, if the arch were as flat as it seemed to be from inspection, the tubercle should have been much lower.

(*b*) *The Tubercle on the Unhealthy Side Higher than on the Healthy Side.*

CASE IV.—M. G., a servant, aged about thirty-five years, was first seen at the Lakeside Dispensary October 19, 1908. The preceding April she sustained a fall of eight feet, landing on the sole of the right foot. The foot became swollen, and has disabled her since. She had been treated at another hospital by having the foot strapped. There had been some relief. The pain was chiefly on the outer side of the foot. Examination showed the right foot permitting almost no motion in inversion, but fairly good motion in dorsal and plantar flexion. The restriction of motion seemed to be chiefly due to mechanical bony interference rather than spasm. Some induration was to be felt under the malleoli and on the dorsum near the cuboid. The tubercle of the scaphoid on the affected side, the right, was one-eighth of an inch below the connecting line, and on the good side three-eighths of an inch below. A Röntgen picture was taken and showed a lesion of the calcaneo-astragalar joint, and signs of impaction in the astragalus with fusion of the calcaneo-cuboid joint.

It seemed that in the original injury the astragalus had been crushed into the os calcis, indirectly injuring the cuboid. This anatomical lesion would explain very well the relative elevation of the tubercle of the scaphoid on the affected side, because the malleoli had been lowered by the crushing of the joint beneath, whereas the position of the scaphoid had not become altered.

CASE V.—M. K., a school boy, aged about twelve years, had infantile paralysis when a child, causing the present deformities. Examination showed the left foot in the position seen in Fig. 9, with the dorsum very high, the toes markedly contracted, the dorsal flexors of the toes prominent, the arch extremely high, the ball of the big toe prominent, and the plantar fascia very tense. This kind of a foot is immediately recognized as a claw foot or non-deforming club foot. The other foot was in equinus, so that it could not be used as a healthy foot for comparison. Measurement of the scaphoid tubercle on the claw foot showed it to be one-fourth of an inch above the connecting line, indicating the extreme elevation of the arch.

The first three cases hardly require additional comment, except to

call attention to the fact that the measurement of the relative depression of the scaphoid tubercle is of considerable help when making comparison with the other foot. In Case IV the reasoning from these landmarks should have carried with it a diagnosis of fracture, even without a Röntgen picture. In Case V the tubercle is actually elevated above the connecting line. It might be thought that, according to our rule, in which it is stated that the foot is deformed according to the deviation of the scaphoid tubercle from the connecting line, there would be confusion in distinguishing such a foot from the "type" foot, but this case brings out the point on which I laid special stress, namely, that other things have to be equal. Here, to be sure, the scaphoid tubercle is markedly elevated, and

FIG. 9.—Case V. Claw foot (non-weight-bearing) from infantile paralysis. Note the elevation of the scaphoid tubercle.

would by itself suggest beauty and function, but the cause of the high elevation, the paralysis (namely, paralysis of the interossei), has also brought about coincidental deformities, that is, contractures. In other words, other things are not equal. There are pathological signs which make up for the height of the arch, and the co-existence of these other pathological signs and symptoms would prevent us from calling such a foot a "type" foot.

II. SYMMETRICAL DEPRESSION OF THE SCAPHOID TUBERCLE. These are the most common group of cases, and, of course, include the ordinary flat foot.

FIG. 10.—Case VI. Flaccid flat foot of moderate degree (non-weight-bearing) in a subject aged ten years. The tubercle is ¾ inch below the connecting line.

FIG. 11.—Case VII. Rigid flat foot, marked (non-weight-bearing). The tubercle is one inch below the connecting line.

Case VI.—F. M., aged eleven years, was seen at the Lakeside Dispensary, March 15, 1908, complaining of pain in both feet for some time. No swelling was noticed. Examination (Fig. 10)

Fig. 12.—Röntgen picture of Case VII. The metal mark shows the position of the scaphoid tubercle.

Fig. 13.—Severe valgus following infantile paralysis. The mid-point represents not the tubercle of the scaphoid but the head of the astragalus.

showed the feet long, lanky, and abducted. There was no stiffness, and the arch was apparently low. The tubercle of the scaphoid was about three-fourths of an inch below the connecting line on each foot without weight-bearing. A diagnosis of moderately flaccid flat foot was made.

CASE VII.—W. H. Five years ago this boy began to limp. His ankles and wrists became swollen. Since then he has stumped about with his feet in the same condition, and has had much pain and swelling. Examination (Fig. 11) showed marked valgus, abduction, and pronation of the ankles, which were swollen and bluish. On palpation the tubercles of the scaphoid were found to be very low, about an inch below the connecting line. The peroneal tendons were tight. There was stiffness in all directions. The patient toed out when he walked. In this case I placed a metal mark on the tubercle and took a Röntgen picture, which proved that this prominent point on the contour was really the tubercle (Fig. 12). A diagnosis of extreme rigid flat foot was made.

Both these cases illustrate common types of flat foot and the relative significance of the depression of the scaphoid tubercle as suggesting the severity of the deformity.

It is, perhaps, worth while to call attention to a possible source of error. In very severe types of flat foot (as from infantile paralysis) the head of the astragalus twists so far inward as to suggest that its prominence is the tubercle of the scaphoid (Fig. 13). Experience and the study of the skeleton, however, will prevent the observer from being misled.

A FURTHER CONTRIBUTION TO MY "SIMPLE" METHOD FOR THE QUANTITATIVE DETERMINATION OF PEPSIN IN A GIVEN GASTRIC JUICE.

BY H. ILLOWAY, M.D.,
OF NEW YORK,
FORMERLY PROFESSOR OF DISEASES OF CHILDREN IN THE CINCINNATI COLLEGE OF MEDICINE AND SURGERY; FORMERLY VISITING PHYSICIAN TO THE JEWISH HOSPITAL, CINCINNATI.

IT is a well-known fact and has been repeatedly demonstrated, by me among others, that the essential factor in the digestion of proteids is pepsin. It is, therefore, of the greatest importance in all cases of chronic gastric disease, especially that form attended with diminished or inhibited secretion of hydrochloric acid, to determine (*a*) whether pepsin is present, and (*b*) if so, whether it is present in normal quantity, in increased or diminished quantity, or (*c*) whether it is altogether wanting.

Various methods have been devised for this purpose, all more or less complicated and not adapted to the needs of the busy practi-

tioner. In 1905 I published[1] my method for the quantitative determination of pepsin found in a given gastric juice. In that article I claimed that my method is the simplest devised, and gives results that are perfectly reliable and in a manner readily and easily interpreted. Since then the application of the method in a much greater number of cases has, to me at least, substantiated all the claims made for it at the time, and has also brought to light some new facts which give it additional value. It is for these reasons that I bring this method once more to the attention of my colleagues.

Since the publication of my first paper two other methods,[2] making the same claims to clinical applicability, have been devised and published. The first is the method of Jacoby and Solms,[3] based upon the fact that a cloudy solution of ricin[4] in dilute hydrochloric acid becomes clear on the addition of a peptic ferment, or a solution thereof.

The technique of the method is as follows: 0.5 gram of the cheap ricin[5] is dissolved in 50 c.c. of a 5 per cent. solution of sodium chloride, and filtered; 2 c.c. of this solution, which is somewhat turbid, is put into each of five test-tubes, and 0.5 c.c. of a decinormal solution of HCl added. The ricin solution becomes at once milky, opaque. The gastric filtrate obtained after an Ewald and Boas test breakfast is variously diluted according to the degree of acidity it is found to possess: The hyperacid, from 1 to 100 to 1 to 10,000; the normal (40 to 60), from 1 to 100 to 1 to 1000. If the gastric juice to be tested is, say, of the normal variety, a 1 per cent. dilution thereof is prepared by adding to 1 c.c of the filtrate 99 c.c. of distilled water. To each of the test-tubes prepared as above, and numbered respectively 1, 2, 3, 4, 5, there is added of the boiled gastric juice (the ferments it contains are destroyed by boiling) to No. 1, 1 c.c; to No. 2, 0.9 c.c.; to No. 3, 0.8 c.c.; to No. 4, 0.5 c.c.; to No. 5, 0 c.c. (none of the boiled filtrate is added). This done, there is further added of the 1 per cent. dilution (unboiled) to No. 1, 0 c.c. (nothing); to No. 2, 0.1 c.c.; to No. 3, 0.2 c.c.; to No. 4, 0.5 c.c.; to No. 5, 1.0 c.c. Each of the test-tubes contains now altogether 3.5 c.c., and we have dilutions of the filtrate, No. 2, 1 to 1000; No. 3, 1 to 500; No. 4, 1 to 200; No. 5, 1 to 100. No. 1, to which no ferment (that is, none of the 1 per cent. dilution) has been added, serves as a control tube. The tubes are now corked and placed in the thermostat and

[1] Einfache Methoden zur quantitativen Bestimmung der vom Magen ausgeschiedenen Enzyme, Archiv f. Verdauungskrankheiten, 1905, xi, 145.

[2] Other methods besides these have been devised since, but they are strictly laboratory methods.

[3] Ztschr. f. klin. Med., vol lxiv.

[4] "Ricin," Kobert, Merck: Exceedingly poisonous agglutinin from the seeds of Ricinus communis L. and closely allied to the bacteriotoxins; yields about 25 to 30 per cent. ash. White Powder.—Sol.: 10 per cent. solution of sodium chloride.—*Caution*: Handle very carefully; small particles in abrasions, eye or nose, may prove fatal; intravenous injections of 3 grains (0.2) are fatal.—Merck's Index, 1907.

[5] An impure preparation made by the Vereinigten Chemischen Farbwerke, Charlottenburg, Berlin.

allowed to remain therein for three hours; then they are taken out and examined to see which dilution has clarified the ricin solution. If a thermostat be not at hand, the procedure may be carried out at room temperature, but then requires a longer time. Having found on repeated examination that, of filtrates of normal acidity, 1 c.c. of a dilution of 1 to 100 (1 per cent.) just cleared the ricin solution in three hours, he called the pepsin content of this 1 c.c. 100 pepsin units, establishing it as his standard for comparison.

Witte[6] has suggested certain modifications in this method. Having found in a series of cases of supposed normal acidity that the pepsin content of 1 c.c. of a dilution of 1 to 100 did not reach 100 pepsin units, he begins his dilutions of gastric juices of normal or slightly lowered acidity with 1 to 50. In cases of marked hypoacidity or anacidity he may use the undiluted filtrate for the first tube and begin his dilutions with 1 to 1. As the HCl element varies greatly in quantity, he would have all gastric juices to be thus examined neutralized with a decinormal solution of potassium hydroxide and then brought to a uniform degree of acidity by the addition of decinormal solution of HCl.

Leaving out of consideration the question of the handling of a substance so fraught with danger, and, furthermore, leaving out of consideration the criticism of the method made by Gross and by Fuld and Levison, namely, that the ricin is full of impurities, and therefore all determinations based upon it are unreliable, a moment's thought will at once demonstrate that it is not a clinical method at all. The solutions and the many dilutions to be prepared, all of which, to give any value to the work, must be made with the greatest accuracy, a minute quantity more or less vitiating the result one way or the other, require more time and more care in their preparation than can be spared by the man in active practice who has a number of gastric juices to examine every day and other duties connected with his work to attend to.

Then, after all that painstaking work, what has the inquirer learned? He has learned that 1 c.c. of a certain dilution of a certain gastric juice is equal to the task of clarifying a certain solution of ricin. Does this reveal to him directly anything as to the digestive capacity of the stomach of the patient from whom the gastric juice was derived? Nothing at all. Only by a long stretch of the imagination and a long mathematical calculation based on figures that are essentially hypothetical (he even does not know the albumin content of the ricin he has used) could he form an idea of the possible digestive capacity of that particular stomach.

The second method is that of Fuld;[7] Levison[8] later published additional details. For his purpose he makes use of a 0.1 per cent. (1 to 1000) solution of edestin, a substance derived from hemp

[6] Berl. klin. Woch., 1908, p. 643.
[7] Münch. med. Woch., 1907, p. 1455.
[8] Amer. Med., February, 1908.

and belonging to the category of the globulins. The edestin is dissolved in a solution of dilute hydrochloric acid having an acidity of 30 (corresponding, as he says, to the free HCl found normally after a test breakfast of Ewald and Boas). Six test-tubes (preferably of a diameter of 1 c.c.) are filled with various quantities of gastric filtrate obtained after an Ewald and Boas test breakfast and diluted according to its degree of acidity from 1 to 100 to 1 to 10 (for anacidity). To each of these tubes 2 c.c. of the edestin solution is quickly added and the contents then allowed to digest for thirty minutes at the room temperature. At the expiration of the time named, strong ammonia is allowed to run down the sides of the tube until a layer is formed above the contents. The tubes are now examined by reflected light against a black background. If digestion is complete, nothing is seen, but if incomplete a distinct white ring of albumin is seen between the two layers—just as in Heller's test for urine.

By finding the test-tube in which a distinct ring ceases to be formed, the point where digestion is complete is determined, and by that the strength of the pepsin content of the given gastric juice. Thus, if the tube with 0.7 c.c. gastric filtrate has no ring, and the next tube with 0.4 c.c. thereof has a ring, it can be said that the gastric juice of that stomach has a pepsin strength of 0.7 in a 1 to 100 dilution. This can be recorded in another way: The dilution of the stomach contents is multiplied by the amount of the edestin solution used and the product divided by the amount of gastric filtrate placed in the tube; thus, 100 x 2.0 (read as 20), divided by 0.7, equals 285+. The pepsin value of that gastric juice is then said to be 285+.

Or the test can be carried out in this way: Instead of the ammonia, a 10 per cent. solution of sodium chloride is added to the tube. This will produce cloudiness proportionate to the degree of digestion that has taken place. The more ample the digestion, the lighter the cloudiness; in other words, the greater the quantity of the solution that will be required to render the contents of the tube cloudy. Thus, at the outset 0.5 c.c. of the salt solution may produce turbidity, while half an hour later it may require 2 c.c., or even more, to produce the same result. In this wise the test can be made with but a single tube.

It can be made in still another way: A 1 per cent. solution of edestin is prepared as above directed. Of this a sufficient quantity (5 or even 10 c.c.) is placed in a test-tube and digested with a not too strongly diluted gastric filtrate. At the expiration of half an hour solid sodium chloride is added, the tube well shaken, and the height of the precipitate measured. Levison says, in further explanation, that "the precipitated albumin very quickly assumes a definite volume, and as it exists under no considerable pressure, its volume is dependent upon the pepsin amount, and allows itself to be read off after standing a short time."

It will be at once apparent to the reader that this method also is not the simple one it is held out to be. All that has been said in criticism, in this one regard, of the method of Solms can be said of that of Fuld, namely, it is too complicated; the making of the principle solution, the dilution of the gastric filtrate, the filling of a number of tubes with minimal quantities which, to give reliable results, must be accurately measured (that it is not an easy matter to measure accurately 0.7 or 0.4 or 0.3 c.c. will be acknowledged by all laboratory workers, and is clearly shown by the numerous instructions given by the writer of the article as to the use of graduated tubes, pipettes with rubber bulb, etc.)—0.05 c.c. too much or too little, and the whole calculation goes wrong.

Then, after all the work has been done, what has the examiner learned? Nothing! His question, whether the gastric juice of a certain patient has enough pepsin content to carry on normally the process of digestion, remains unanswered.

A method to be clinical, that is, adapted to the needs of the man of large practice with not much time to spare, must be possessed of the following qualifications: (1) It must be simple in its technique, and every step thereof definite and accurately measured; and (2) it must give a result at once apparent, at once understood, without the necessity of long and complex calculations or of the translation by way of the imagination of hypothetical figures into those of a supposed actuality.

Of all the methods hitherto described, only the one devised by me measures up to this standard. It is as follows:

(*a*) Ten centigrams (exact weight) of egg albumen (white of egg[9]) is coagulated in the following manner: The egg is put into a pot of cold water, which is then covered with a lid and set on to cook. It is allowed to cook for ten minutes after the water has begun to boil—in all, twenty minutes from the time it was put on. The egg is then taken out and allowed to cool, either by just setting it out in a saucer or by putting it into cold water. Furthermore, to imitate the usual way in which food is or should be put into the stomach, the segment of albumen is divided into two parts. (Observation has shown—just as it has long been known clinically—that when it is thus subdivided the gastric juice can act upon it more quickly. The action being from all sides, it is naturally more effective when there are eight sides of attack for a given quantity than when there are but four.) (*b*) The two parts of coagulated egg albumen are put into 10 c.c. of the gastric filtrate from the stomach contents extracted one hour after an Ewald and Boas test breakfast; and (*c*) they are then placed in the thermostat, which is kept at 38° C. The time in which the 10 centigrams is digested entirely, partially, or not at all, will give us a correct idea as to the status of the pepsin secretion in the case under examination.

The results obtained with this method are well shown in the following tables:

[9] Hens' eggs only.

TABLE I—RAPID DIGESTION (less than the normal period.)

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I F.	N. K.	42	62	Hyperacidity	2.23	
II M.	A. A.	38	48	Hyperacidity, subacute gastritis, constipation	3.00	15 centigrams in 10 c.c. filtrate digested fully in 5¼ hours.
III M.	F. H.	66	99	Hyperacidity, constipation	3.00	15 centigrams in 10 c.c. filtrate digested fully in 3½ hours.
IV M.	R. M.	56	77	Hyperacidity	3.10	
V M.	S. B.	62	77	Hyperacidity, constipation	3.10	
VI F.	M. B.	12	28	Deficient HCl; in the climacteric	3.15	Eats too heavy food. Her teeth are bad, and she cannot masticate well.
VII M.	A. E.	50	68	Hyperacidity, constipation	3.21	
VIII M.	J. K.	47	69	Eats too much and too heavy food	3.26	Adenoids and obstructed nose.
IX M.	A. K.	71	102	Hyperacidity and constipation	3.29	
X F.	B. C.	38	64	Hyperacidity, constipation	3.30	6 centigrams in 5.5 c.c. filtrate digested in 3½ hours.
XI F.	Y. G.	35	58	Constipation, inflamed hemorrhoids	3.30	
XII M.	J. F.	No reaction to phlor. gluc. v.	24	Constipation, colics, deficient HCl	3.30	With the addition of HCl c. p., gtt. ii. Without the acid at the end of 11 hours, the edges only slightly thinned.
XIII M.	J. L.	46	67	Constipation, hyperacidity	3.40	
XIV F.	E. A.	20	54	Constipation, dyspepsia	3.42	
XV M.	A. B. F.	40	57	Hyperacidity, constipation	3.49	His father suffered from hyperacidity, and finally died in an attack of hematemesis.
XVI M.	B. I.	29	58	Constipation	3.49	
XVII M.	B. G. C.	70	115	Hyperacidity	3.50	15 centigrams in 10 c.c. filtrate in 3 hours 50 minutes all digested except one small particle. That remained even after 4 additional hours.
XVIII M.	J. P.	39	54	Neurasthenia, constipation, hyperacidity	3.50	Three months later similar trial; 10 centigrams in 10 c.c. filtrate digested in 3 hours and 30 minutes.
XIX M.	N. W.	63	85	Hyperacidity, constipation	3.56	
XX M.	E. W.	41	59	Neurasthenia, hyperacidity	4.00	
XXI F.	S. M.	47	69	Hyperacidity, constipation	4.00	
XXII M.	E. M.	19	37	Atony of stomach	4.00	Lives in a warm climate Eats food that is too heavy.
XXIII M.	L. R.	56	87	Constipation, hyperacidity	4.00	
XXIV F.	M. D.	22	54	Subacute gastritis	4.00	Had a gastro-enterostomy made on her before I saw her.
XXV M.	G. W. F.	39	71	Hyperacidity	4.09	
XXVI M.	I. B.	17	31	Constipation, slight deficiency in HCl	4.12	
XXVII F.	S. W.	27	57	Subacute gastritis	4.15	
XXVIII M.	L. B.	22	46	Constipation, secondary atony of stomach	4.15	
XXIX M.	A. W.	41	64	Hyperacidity, constipation neurasthenia	4.15	

TABLE I.—Continued.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
XXX M.	I. C.	48	79	Hyperacidity, constipation	4.19	
XXXI M.	A. M.	50	61	Tabes, (beginning of) hyperacidity	4.20	
XXXII M.	M. A.	45	61	Hyperacidity, constipation	4.20	
XXXIII F.	S. H.	08	24	General debility, neurasthenia	4.20	
XXXIV M.	G. M.	38	64	Hyperacidity	4.25	
XXXV M.	Ph. M.	29	50	Constipation, uric acid gravel	4.25	
XXXVI F.	Mrs. B.	28	55	Hyperacidity	4.29	Determined from the symptoms, which are the same as in the most marked cases.
XXXVII F.	F. S.	28	51	Neurasthenia, constipation	4.30	
XXXVIII M.	D. H.	53	81	Hyperacidity, constipation	4.30	
XXXIX M.	M. S.	25	50	Subacute gastritis	4.30	
XL F.	D. B.	20	40	Subacute gastritis, diarrhœa	4.30	
XLI M.	N. P.	29	61	General debility, constipation	4.30	Taught school and studied law.
XLII M.	S. B.	22	45	Neurasthenia	4.54	Repeated a month later. Same results.
XLIII M.	H. C. B.	45	64	Hyperacidity, constipation	4.35	
XLIV M.	B. M.	19	34	Dyspepsia, constipation	4.40	
XLV M.	H. R.	36	59	Hyperacidity	4.40	
XLVI M.	H. G.	23	81	Hyperacidity, subacute gastritis	4.45	
XLVII M.	S. Gr.	24	58	Stricture of pylorus	4.45	

As already stated elsewhere,[10] and for the reasons there given, I fixed upon 10 centigrams of albumen in 10 c.c. of filtrate, and five to five and one-half hours (at the farthest) to be taken as the normal standard for both quantity and time. Any deviation from this standard in either direction could then be readily recognized, and this deviation should then be, as it has been in all my cases, in harmony with the clinical history. That this method is correct for both quantity and time was borne out by the first series of cases reported,[11] and is fully confirmed by the tables here presented.

Table I shows a deviation downward. The 10 centigrams is digested more rapidly than usual, showing a greater digestive force in those particular gastric juices. This was proved in some of the cases (in those in which sufficient of the filtrate remained over for further examination) by a subsequent examination with 15 centigrams of the albumen to 10 c.c. of filtrate. In my first series of cases (coming under this same head), in all the instances in which a second examination with 15 centigrams was made the time required for the digestion of the greater quantity was the full normal time, five and one-half hours. In the present series we have three cases in which this examination

[10] Archiv. f. Verdauungskrkh., loc. cit.

[11] Loc. cit.

was made. In Case II the time required for the digestion of the 15 centigrams was five and one-quarter hours, the normal time. It indicated that in this case the pepsin in the gastric juice was augmented by 50 per cent. beyond the normal.

TABLE II.—DIGESTION IN NORMAL PERIOD.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I F.	L. H.	32	53	Constipation, hyperacidity	5.00	
II M.	M. C.	45	67	Hyperacidity, subacute gastritis, constipation	5.00	Suffers much with cramps.
III M.	M. J. C.	17	44	Constipation, dyspepsia	5.00	
IV F.	R. H.	24	48	Constipation; impaired respiratory activity	5.00	Nose very much obstructed.
V M.	S. B.	25	40	Stricture of œsophagus	5.00	
VI F.	L. C.	21	51	Constipation, colics	5.00	
VII M.	L. G.	26	60	Constipation	5.00	The constipation due to a wrong diet.
VIII F.	L. T.	19	40	Constipation	5.02	
IX M.	I. S.	84	98	Hyperacidity	5.03	
X M.	C. B.	35	65	Hyperacidity	5.05	
XI M.	J. H.	59	84	Hyperacidity, constipation	5.05	
XII M.	M. W.	26	64	Colics	5.09	
XIII F.	A. M. H.	28	56	Dyspepsia	5.10	
XIV M.	W. S.	25	49	Subacute gastritis, gastrosuccorrhœa	5.10	Subsequently obstruction of bile duct and jaundice.
XV M.	M. A.	46	62	Bright's disease	5.12	
XVI M.	C. N.	25	48	Hyperacidity, dyspepsia, constipation	5.12	
XVII M.	S. A. V.	37	53	Hyperacidity, subacute gastritis	5.13	
XVIII F.	N. D.	34	46	Constipation, tendency to hyperacidity	5.18	
XIX F.	Mrs. Z.	30	69	Neurasthenia	5.20	
XX F.	F. G.	30	45	Bad mastication, bad teeth, constipation	5.20	
XXI M.	A. B.	33	59	Tendency to hyperacidity, irritability of stomach, nervous	5.24	
XXII M.	S. R.	41	61	Hyperacidity	5.24	Nervous temperament.
XXIII M.	M. B.	34	59	Hyperacidity, constipation	5.30	
XXIV F.	F. R.	13	69	Constipation, dyspepsia	5.30	
XXV M.	F. B.	24	45	Rheumatism of stomach, subacute gastritis	5.30	Tendency to hyperacidity.
XXVI M.	M. G.	44	76	Hyperacidity	5.30	Much coffee.
XXVII F.	A. R.	19	39	Constipation, deficient HCl, nervousness	5.30	Leads an irregular life.
XXVIII M.	C. J. L.	055	25.5	Deficient HCl, irritability of intestinal tract	5.30	
XXIX M.	A. E.	30	52	Subacute gastritis	5.30	
XXX M.	S. J. P.	59	81	Hyperacidity, slight subacute gastritis	5.30	

In Case III the 15 centigrams was digested in three and one-half hours, showing that in this case the quantity of pepsin secreted was greater even than in Case II, possibly double the normal. I regretted

TABLE III.—DELAYED DIGESTION (more time than the normal period required).

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I M.	M. P.	31	51	Constipation, tendency to hyperacidity	5.50	At the time of examination a question as to whether he had had a hepatitis shortly before or not.
II F.	M. M.	28	44	Constipation, secondary atony of stomach	6.00	
III F.	L. P.	22	51	Chronic diarrhœa	6.00	
IV M.	M. L. R.	26	71	Hyperacidity, constipation, angina pectoris	6.10	
V F.	N. B	03	37	Chronic gastric catarrh	6.10	Has lost very much flesh.
VI M.	M. S.	25	50	Hyperacidity, pseudo-angina pectoris	6.10	The symptoms pointed thereto (hyperacidity) and the dietary directions for this same brought rapid relief.
VII F.	L. K.	05	26	Defective gastric juice, costive	6.20	
VIII F.	F. D.	10	32	Defective gastric juice, constipation	6.20	
IX F.	P. W.	14	27	Slight catarrh of the stomach	6.20	
X M.	F. D.	13	45	Defective gastric juice, constipation	6.30	
XI M.	J. C. L.	10	29	Defective gastric juice, constipation	7.00	
XII M.	L. M.	45	65	Hyperacidity, constipation	7.03	
XIII F.	L. K.	05	28	General impairment of the digestive organs reflexly from ailments of the genital tract.	7.15	
XIV F.	B. K.	No reaction to Congo, none to ph. gluc. v.		Chronic gastric catarrh, tendency to epileptic seizures.	10.20	

very much that for lack of filtrate I could not carry the examination farther, to 17.5 centigrams, and if this was digested in less than the normal time, to 20 centigrams.

In Case XVII the greater part of the 15 centigrams was digested in three hours and fifty minutes; a small particle remained over and was still there and as large after four hours more in the thermostat. This demonstrated that the digestive capacity of these 10 c.c. of filtrate was beyond 10 centigrams, but not quite up to 15. Here also the lack of filtrate prevented a reëxamination with a less quantity, namely, 13 centigrams (the particle remaining over in the second examination I judged to be about 2 centigrams).

TABLE IV.—INSUFFICIENCY OF PEPSIN (in all these cases the heating was continued for 4 or 5 hours more after the time named, but no further digestion was effected).

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion and remarks.
I M.	F. R.	43	69	Hyperacidity, constipation	In 3 hours 39 minutes all digested except two or three minute particles.
II F.	B. B.	23	69	Stricture of pylorus (benign)	In 4 hours all digested except a number of minute particles.
III M.	J. H. C.	47	72	Hyperacidity, constipation	In 4 hours 10 minutes all digested except one fine scale. At the end of 7 hours scale still present.
IV M.	S. L. J.	48	71	Hyperacidity, costiveness	In 4 hours 30 minutes all digested except three minute particles.
V F.	L. S.	16	37	Deficient HCl, constipation	In 4 hours 40 minutes all digested except a few minute particles.
VI F.	Mrs. H.	14	51	Constipation, flatulence	In 5 hours all digested except two minute particles.
VII M.	B. S.	73	93	Hyperacidity	In 5 hours all digested except one particle.
VIII M.	M. C.	41	60	Hyperacidity, constipation	In 5 hours all digested except two minute particles.
IX F.	L. S.	08	27	Defective gastric juice, constipation	In 5 hours all digested except one minute particle.
X F.	L. J.	15	38	Gastralgia, constipation, defective gastric juice	In 5 hours all digested except a few minute particles. Five centigrams in 10 c.c., all digested in 3 hours 30 minutes.
XI M.	L. B.	32	58	Hyperacidity, constipation	In 5 hours 7 minutes all digested except two or three particles.
XII F.	V. B.	21	51	Cystic tumor of right ovary	In 5 hours 7 minutes all digested except four or five minute particles.
XIII M.	J. G	48	64	Hyperacidity	In 5 hours 25 minutes all digested except one faint scale.
XIV M.	H. G.	77	96	Hyperacidity, constipation	In 5 hours 40 minutes all digested except a few minute particles.
XV F.	A. C.	12	34	Defective gastric juice, constipation	In 6 hours all digested except three or four particles.
XVI F.	A. R.	12	42	Defective gastric juice, constipation	In 6 hours all digested except three or four minute particles
XVII F.	R. L.	27	50	Status epilepticus	In 6 hours 29 minutes all digested except two minute particles
XVIII F.	H. H.	06	21	Defective gastric juice, constipation	In 6 hours 30 minutes all digested except one small piece, thinned at edges only.
XIX M.	W. K.	55	70	Hyperacidity	In 6 hours 35 minutes all digested except two small particles.
XX M.	J. M.	53	72	Hyperacidity, nervous	In 6 hours 41 minutes all digested except two particles.
XXI M.	D. U.	41	65	Neurasthenia, hyperacidity	After 7 hours a scale of one segment (about one-third of it) remained undigested.
XXII F.	F. J.	10	58	Defective gastric juice, atony of stomach, hyperesthesia of intestinal mucous membrane	After 7 hours 13 minutes a large part of the one segment (though borders were very much thinned down) and a large scale of the other segment remaining over.
XXIII F.	Mrs. H.	13	28	Defective gastric juice, constipation, irregular eating, rich food	After 7 hours 15 minutes a very small particle and several minute ones over.
XXIV M.	L. D.	11	32	Defective gastric juice, slight catarrh of stomach	After 7 hours 30 minutes a number of minute particles still present.
XXV F.	J. C.	27	51	Chronic ovaritis, chronic appendicitis	After 7 hours 45 minutes a number of minute particles still present.
XXVI F.	J. S.	32	49	Atony of stomach	After 7 hours 48 minutes two small scales still present.
XXVII M.	M. R.	29	43	Constipation	After 8 hours several minute particles still present.
XXVIII M.	J. F	34	63	Hyperacidity, atony of stomach second degree, atony of bowels	After 8 hours one small particle and several minute ones still over. (Test repeated; same result.)

TABLE IV.—Continued.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion and remarks.
XXIX F.	Miss L.	14	43	Defective gastric juice, atony of stomach	After 8 hours all digested except one thin scale.
XXX M.	S. J. P.	26	41	Gastralgia	After 8 hours several small particles still present.
XXXI F.	U. J. K.	02	..	Chronic gastric catarrh	After 8 hours 30 minutes 2.5 centigrams over.
XXXII M.	W. A. J.	58	77	Hyperacidity	After 8 hours 40 minutes two or three particles still present.
XXXIII M.	J. B.	43	78	Hyperacidity (bad teeth, bad mastication)	After 8 hours 43 minutes four small particles still there.
XXXIV M.	S. P.	21	47	Gastralgia	After 9 hours a number of minute particles still present.
XXXV M.	S. E.	17	49	Sarcoma of intestines	After 10 hours a scale of each segment left.
XXXVI F.	R. B.	16	26	Gastralgia, constipation	After 10 hours a minute particle still present.
XXXVII M.	J. W.	21	37	Beginning gastric catarrh	After 10 hours 30 minutes two or three minute particles still present.
XXXVIII	B. Th.	40	52	Hyperacidity, constipation	After 11 hours a minute scale still present.
XXXIX	A. K.	09	39	Defective gastric juice, constipation	After 11 hours two or three minute particles still present.
XL F.	J. R. W.	10	41	Defective gastric juice	After 12 hours three minute particles still over.
XLI F.	B. K.	10	28	Chronic gastric catarrh	After 13 hours four minute particles still present.

TABLE V.—VERY LITTLE DIGESTION OR NONE AT ALL.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Time.
I F.	C. T.	No reaction to Congo	..	Chronic gastritis	10 centigrams in 10 c.c. filtrate + gtt. ii HCl c.p. After 9 hours unaffected.
II M.	E. N.	Reaction: blue litmus + Congo — resorcin —	..	Chronic gastro-intestinal catarrh	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl gtt. ii c.p. After 11 hours digested down to one large scale and several small particles (in all about one-third, left.
III F.	S. W.	Reaction: blue litmus — Congo —	..	Cirrhosis ventriculi	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl c.p. gtt. ii, no effect.
IV F.	S. R.	Reaction: Congo + filtered Congo — ph. gluc. v. —	18	Neurasthenia, constipation	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl c.p. gtt. ii, digested in 6 hours.
V M.	N. S.	Reaction: blue litmus + Congo —	..	Nervous prostration	Without HCl, no effect; with HCl, digested all but one particle in 6 hours.
VI M.	A. St.	Reaction: blue litmus + Congo —	..	Cirrhosis ventriculi	10 centigrams with HCl gtt. ii at end of 5 hours untouched.
VII M.	H. W.	Reaction: blue litmus + Congo —	..	Cirrhosis ventriculi	10 centigrams with HCl at end of 10¾ hours untouched.
VIII M.	N. R.	Reaction: blue litmus + Congo —	..	Chronic gastric catarrh	10 centigrams without HCl after 10¾ hours no digestion at all. With HCl at end of 10¾ hours one large scale and a few particles over, altogether about 3 centigrams left over.
IX F.	S. F.	Reaction: blue litmus + Congo —	..	Chronic gastric catarrh	Without HCl, unaffected. With HCl at end of 8 hours, one segment slightly thinned at edges.
X M.	S. D.	0.1	2.2	Chronic gastric catarrh	Without HCl after 9 hours and 7 minutes, the four borders of each segment thinned down, translucent almost. With HCl, all digested in 9 hours and 7 minutes, except 2 small fine scales.
XI F.	J. V.	Filtered. No reaction to ph. gluc. v.	3.0	Constipation, neurasthenia	Without HCl, not affected. With HCl at end of 5 hours and 7 minutes all digested except two minute particles.

The points of special interest in this table are the indications (*a*) that in many cases of hyperacidity, perhaps in the greater number, there is also an increased secretion of pepsin,[12] and (*b*) that it is not by the excess of acid that the digestion is hastened. Thus, in Case XIV, with a degree of acidity, both free and combined, considerably lower than many of the others that follow, the digestive act was complete in much less time than was required, say, in Case XVII, with more than three times the quantity of free acid and more than double the quantity in the total acidity. The same can be seen in a number of other cases in this same table, and it is very clearly shown when comparisons are made with some of the cases of hyperacidity (Cases IX, XI, and XXVI) of Table II, and most strikingly so when the comparisons are made with some of the cases of hyperacidity of Table III (Cases IV and XII) and of Table IV (Cases IV, VII, and VIII).

There is yet something more that this table seems to disclose. It is the possibility that just as there may be an increased secretion of HCl without a corresponding increase in the secretion of the pepsin, so there may be an increased secretion of pepsin without any corresponding increase in the secretion of HCl. Cases XIV, XXII, XXVI, and XXXIII seem to point thereto.

In Table II the cases all conform to the standard in quantity and time. Here also we have a very interesting observation, the corollary of that noted above, namely, that there may be an increased secretion of HCl without a corresponding increase in the pepsin.[13] The patients with hyperacidity of Table I had, with all their complaints, a fair to good appetite, and could, when properly directed as to diet, eat again four hours after a hearty meal; those of Table II did not have that early hunger—in fact, some of them complained of poor appetites. In some of the cases of this table the greater part of the 10 centigrams was digested in four hours or a little over; still, as long as a speck remained, the act was not counted complete.

Tables III, IV, and V show us the deviations upward, there being a deficiency in the quantity of pepsin secreted. In Table III we note a deficiency; the time required for a completion of the process of digestion is more or less greater than the normal time. Nevertheless, the deficiency can be but slight, as there is still enough pepsin secreted to digest completely the test quantity. In Table IV the deficiency is greater, and part of the test quantity remains undigested. How much this is can be readily estimated either by simply judging it by the eye, which is rather unscientific, or by the more accurate method of removing it from the beaker to a filter and weighing it.

Still another fact of great interest is presented to us by these tables, namely, that we may have hyperacidity associated with a deficient

[12] This was first definitely demonstrated by my tables.

[13] Ibid.

secretion of pepsin, from the minimal deficiency of Table III, causing only a protraction of the act of digestion, *i. e.*, requiring a longer time for its completion, to the larger one of Table IV, entailing a marked diminution of the digestive capacity of that gastric juice.

In Table V we observe a total or almost total absence of pepsin and pepsinogen. Here we have a further demonstration of the fact that the HCl constituent alone does not effect digestion. In Cases I, III, VI, and VII pepsin and pepsinogen were altogether wanting, and the addition of sufficient HCl (c. p.) to give free acid 22 did not in any way change the result.

To summarize, it may be claimed for this method:

1. That it is simple and eminently practical.

2. That from the simplicity and exactness of its preliminaries it is well adapted for comparative study.

3. That it is directly demonstrative, in that the examiner has a direct ocular demonstration, as it were, of the digestive capacity of a given gastric juice, and can thus at once, without any long mathematical calculations, which not infrequently have only a fictitious basis, determine whether in the case under examination there is:

Hyperpepsinia: Requiring only three to four hours for digestion. Not necessarily in a pathological sense, only to indicate a secretion of pepsin greater than usual, which may, however, be normal for that case.

Normal pepsinia: Requiring only five to five and one-half hours (the normal period) for digestion.

Hypopepsinia: Requiring more than the normal time for digestion (deficiency in the pepsin secretion).

Apepsinia: No digestion at all (total absence of pepsin and pepsinogen).

TABLE VI.—DIGESTION IN LESS THAN THE NORMAL PERIOD. Less than 10 c.c.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Quantity.	Time.
I M.	E. G.	45	85	Hyperacidity	4 centigrams to 4 c.c. filtrate	3.55
II F.	P. K.	11	22	Deficient HCl, constipation, nervousness	5 centigrams to 5 c.c. filtrate	3.55
III M.	J. L.	42	74	Hyperacidity, intestinal colic	5 centigrams to 5 c.c. filtrate	3.30
IV F.	B. C.	38	64	Hyperacidity, constipation	6 centigrams to 5.5 c.c. filtrate	3.30
V M.	J. St.	65	92	Hyperacidity, constipation	7 centigrams to 7 c.c. filtrate	3.35

But a mathematically still more closely defined result can be obtained with this method. In the course of this work it occurred to me, and the reasons therefor are self-evident, that possibly each of the 10 c.c. possessed the power of digesting individually 1 centi-

gram of the white of egg above described. A number of trials were made, and the results proved this to be so.

Additional confirmation was afforded by the results obtained in some other cases:

In Case X of Table IV the 10 c.c. were not able to digest the 10 centigrams entirely; a few minute particles remained over, and allowing the beakers to remain in the thermostat for four or five hours more than the normal time did not change the result; but 5 centigrams was digested by 10 c.c. in three and one-half hours, two hours less than the normal time. It was clearly apparent, therefore, that though the 10 c.c. was not quite equal to 10 centigrams (in other words 1 c.c. to 1 centigram), it was more than equal to 5 centigrams (or 1 c.c. to more than 0.5 centigram). If more filtrate had been left the examination could have been continued until the exact limit for quantity had been reached; judging by what was left over on the first trial, I should say that this would have been about 9 to 9.5 centigrams.

F. A. G., reaction to blue litmus +	6 centigrams in 6 c.c. after 9 hours 15 minutes, edges only slightly thinned.
reaction to Congo +	6 centigrams in 6 c.c. + HCl (c. p.) gtt. ii. Complete digestion in five hours.
Filtered, reaction to Congo —	

Here, when we have the pepsin activated by the addition of HCl, we get a striking verification of our proposition. It can, therefore, be said that the normal quantity of pepsin in the ordinary normal stomach is equivalent to the digestion by each cubic centimeter of the gastric juice of 1 centigram of white of egg, prepared as above described, in from five to five and one-half hours.

ADENOFIBROMA OF THE MALE BREAST.

By R. T. Woodyatt, B.S. M.D.,
of Chicago.

(From the Pathologic-Anatomical Institute of the University of Strassburg, Prof. Chiari, Director.)

In March, 1907, there was sent to the laboratory a tumor mass which had been extirpated from the breast of a boy. This was put into my hands for study by Prof. Chiari. It had a fibro-epithelial structure corresponding to that of ordinary adenofibroma, such as may occur in the female breast. In attempting to ascertain the frequency of such growths in the male breast some difficulty was encountered, because of the uncertainty which still prevails in the literature

regarding the use of terms covering a group of affections of the male breast, all of which are fibro-epithelial in structure. Thus the terms adenofibroma, gynecomastia, adolescent tumor, mastitis, etc., are applied by different writers to the most variable affections. This seems to be due to a lack of data concerning the finer structure of the affections in question, a lack due in its turn to scarcity of pathological material, because all of these affections are uncommon and of such nature that they seldom demand surgical treatment.

A second case like the first one appeared in one of the surgical clinics a few weeks later, and Prof. Chiari then unearthed from the museum specimens representing five more cases of the same general group. With this rather unusual material at hand, it was thought worth while, in view of the afore-mentioned considerations, to record the findings and make them the basis for a comparative study of the members of the group of non-malignant fibro-epithelial tumors which affect the male breast.

CASE I.—(The history was provided by the physician in charge of the case, Dr. Schaller, of Barr, in Alsatia.) Alphonse S., aged fifteen years, a gardener's apprentice, had always been well until one year ago. At this time he noticed a lump the size of a bean under the left nipple, and a few weeks later a similar enlargement under the right. Both were painful and grew quite rapidly until they reached the size of hazelnuts. After four to six weeks they disappeared spontaneously. Toward the end of January, 1907, about a year later, a new tumor appeared under the left nipple. This increased steadily in size, becoming more and more painful until the patient sought the advice of a doctor. He complained then of pain in the left breast which was increased when pressure was made over the areola. There was no history of traumatism.

Physical Examination. The patient is a very thin, lightly built, but healthy looking boy. All organs are normal except the breast. Under the left nipple is an elongated mass, 6 to 8 cm. long and 3 to 4 cm. broad, its long axis extending toward the axilla. Its consistence is firm, its surface somewhat knobbed; it can be moved freely on the underlying structures. The overlying skin is not adherent. The papilla itself, however, is fixed to the mass. There is marked tenderness on pressure. The right breast is normal. A potassium iodide salve was prescribed. February 26 (four weeks later): The breast is more painful. Two bean-sized glands are now palpable in the axilla of the affected side. A compression bandage applied. March 11: The tumor has increased somewhat in size and is still more painful. Extirpation of the mass together with the nipple under local anesthesia. Rapid healing. March 19: The patient reports that he is free from symptoms. Since this time there has been no recurrence.

Macroscopy. The tissue sent to the laboratory in alcohol and formaldehyde consists of a flat, dense, whitish disk (2.5 to 3.0 cm.

broad, 0.5 to 0.8 cm. thick. Its surface is smooth but somewhat knobbed, with all evidence to show that it had been sharply defined from the surrounding tissues. Its cut surface is white with small punctate or linear markings corresponding to gland ducts. These radiate downward and outward from the region of the papilla to the periphery of the disk.

Microscopic examination shows a predominance of fibrous stroma arranged in broad trabeculæ, which support in their interspaces glandular structures (Fig. 2). The latter consist of gland ducts cut in various directions, and their acinus-like terminals. The large ducts are nearly all dilated and show great irregularity of form due to projections of the walls into the capacious lumens. These projections are sometimes purely epithelial; more often they appear to be caused by invaginations of the surrounding stroma. Where both processes combine, true papillary structures are produced (as pictured in Fig. 4 from Case II). The lumens are generally empty. A few contain scattered epithelial cells, clear serum-like fluid, or detritus. Lining the lumens four to six irregular layers of epithelial cells may often be found, while at times, as above noted, solid epithelial projections occur, leaving no doubt that genuine proliferation of these cells has taken place. The ducts after three to four bifurcations terminate in groups of end-sacs analogous to those seen in the breast of a non-parous girl. They vary in number from two to eight in a group. The epithelium everywhere rests on a basement structure which is usually thickened. This membrane is not always "structureless" as Kölliker[1] observes for normal glands. Favorable sections show plainly that it is made up of very long, narrow cells laid in imbricated series and apparently analogous to the layer of myo-epithelium which occurs in sweat glands. Outside of the basement structure comes the fibrous stroma. This is moderately vascular and well supplied with nuclei which stain deeply. (For comparison of stroma with that of the normal gland of a fifteen-year-old male, see Figs. 1 and 2.) In the zones immediately adjacent to the gland structures one notices even greater cell richness and vascularity than elsewhere, also occasional plump cells with large oval nuclei like young connective tissue cells. (These are pictured from Case II, in Fig. 4, D.) The close proximity of these to the thickened basement structure and the occurence of large cells in the latter make it difficult to tell where stroma leaves off and basement structure begins. In hemalum- or hematoxylin-stained specimens the periglandular zones are rendered conspiciously blue owing to the presence there of a fluid which takes that color. Blue zones are especially marked about the terminals and finer ducts of the glands where the parenchymatous parts are invested in loose-meshed

[1] Beiträge zur Kenntnis der Brustdrüse. Verhandl. der physikalisch-medizinischen Gesellschaft in Würzburg, 1880, xiv.

areolar tissue mantels which fill the interspaces between the firm stroma and the epithelial structures. The acini and fine ducts appear then to be surrounded by myxomatous tissue. The fluid, however, gives no characteristic color reactions when stained for

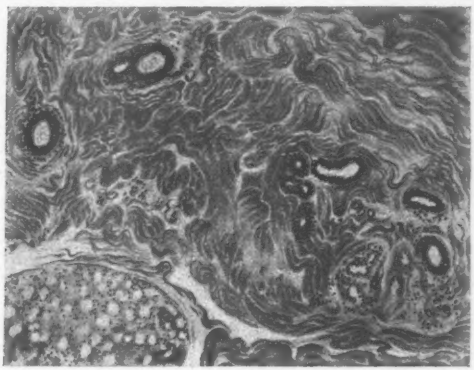

FIG. 1.—Normal breast from a fifteen-year-old boy dead of pneumonia. To show the nature of the normal stroma and epithelium. (Zeiss ocular 4, objective AA.)

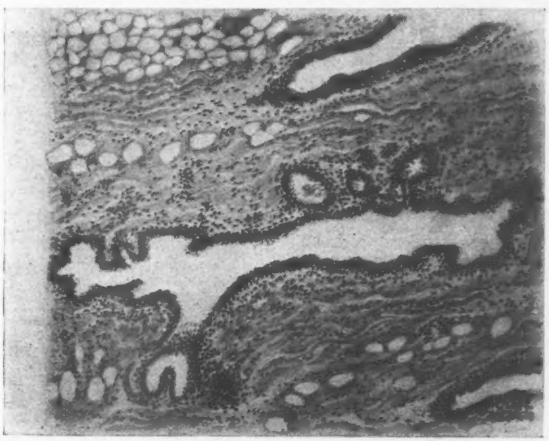

FIG. 2.—Case I. Shows dense rich stroma and proliferated epithelium. (Zeiss ocular 4, objective AA.)

mucin or for fibrin. In adenofibroma of the female breast Ribbert refers to it as more in the nature of œdema. There are no collections of round cells. Elastic tissue is not demonstrable except in the bloodvessels. Fat tissue occurs peripherally in narrow islands

parallel to the fiber course. Centrally one sees single fat cells or small collections of them.

DIAGNOSIS. The following affections require consideration:

I. Simple inflammation (mastitis).
II. Gynecomastia.
III. Neoplasm (adenofibroma).
IV. Adolescent tumor ("mastitis adolescentium," etc.)[2]

I. MASTITIS. There are no evidences of simple acute inflammation. There is no marked vascular engorgement, no cellular nor fibrin-containing exudate. The mass as a whole, was sharply circumscribed. The only feature suggesting inflammation in any way was the enlarged axillary gland. The great difficulty that may arise in trying to differentiate chronic inflammation from tumor in the breast in general is recognized. It has been emphasized by Billroth,[3] Borst,[4] Ribbert,[5] Fabian[6] and others. In this case nothing points unequivocally toward inflammation in the ordinary sense.

II. GYNECOMASTIA. This term, originated by Paul d'Egine, which has been loosely applied to nearly every disease to which the male breast is heir, refers literally to cases in which a male possesses a breast or breasts like those of a woman. Lack of pathological material for accurate study due to the rarity of the affection[7] and its mild nature, which only exceptionally demands surgical treatment, has been responsible for confusion as to its relationship to other diseases of the male breast and misconception as to the degree of real similarity which can exist between male and female breasts in general. Before proceeding with the differential diagnosis, I shall attempt to define clearly what the term gynecomastia properly embraces.

It was at one time supposed and still often stated that such glands can secrete milk.[8] The case of a man who suckled his own child continually recurs in the literature. The weight of scientific evidence is all against such occurrences. No case is on record in which the secretion of milk has been verified by chemical tests,[9] the reports of

[2] Peculiarities of the normal male breast capable of confusion with disease processes are thoroughly treated by Gruber, Luschka, Kölliker, and Langer.

[3] Die allgemeine chirurgische Pathologie und Therapie, 1893, 902.

[4] Die Lehre der Geschwülste. [5] Lehrbuch der speziellen Pathologie, 1902, 564.

[6] Die Bindegewebshyperplasie im Fibrom und im Fibroadenom der Mamma, Arch. f. klin. Chirurgie, lxv.

[7] According to Duplay and Reclus, only one case was found by Peuch in the course of the examination of 13,000 conscripts.

[8] Lydston, Jour. Amer. Med. Assoc., December 12, 1908, makes this statement again apropos of an article by Gellhorn concerning Abnormal Secretions from the Mammary Glands in Non-pregnant Women (same Journal, 1908, li, 1839).

[9] A simple chemical test is to demonstrate the presence of lactose. The poorest true milk contains a percentage of lactose not approached by any other body fluid. If the de-albuminised fluid reduces the ordinary copper solutions, but fails to show fermentation with yeast, lactose is probably present. Further proof of lactose would be afforded by precipitation of phenyl-lactosazon in the usual manner of osazon preparation, separation of the crystals, and determination of their melting point.

milk secretion having arisen through the not infrequent observation of whitish fluids expressible from the nipple which on exact examination have proved to be cystic, purulent, or serous. Further, no careful microscopic examination of the male breast has ever demonstrated acini of the type which occur in a breast which has functionated. A true resemblance between a mature female mamma and that of a male, therefore, does not exist. However, the finer structure of a virgin female breast differs little from that of the male. Let a male breast develop sufficiently in size, and it may truly resemble a nulliparous female breast.[10] Such an enlargement constitutes gynecomastia. It may occur as one manifestation of a general feminine tendency, and be accompanied by other anomalies, such as high voice, female distribution of hair, etc.; it may be associated with cryptorchism, hypospadias, hermaphroditism, and many other anomalies of the genitalia; it may follow removal of the testicles even late in life, or occur in the course of diseases, such as carcinoma, which destroy these organs; or, finally, it may be an apparently isolated anomaly occurring in otherwise healthy men of strong masculine habitus. It may be unilateral or bilateral.

The histology of such glands has been accurately studied in only a few cases (Volkman,[11] Stieda,[12] Israel,[13] Scheiber[14]). Apart from slight differences in their findings, which might have been due to differences in the age of the disease or to the presence or absence of slight superposed pathological processes, such as inflammations, these authors agree in assigning to such glands the structure of hypertrophic male breasts. They are anomalies of growth.

It must be remembered that a hypertrophic male breast is subject to disease. Owing to the nature of a man's pursuits it is more likely to suffer from traumatism than a female breast of the same size. Failure to discriminate between "healthy" and "diseased" gynecomastias has led to many doubtful statements regarding their characteristics, both pathological and clinical. A hard, dense breast is never pure gynecomastia, nor are simple gynecomastias "exquisitely tender" nor painful any more than normal female breasts. Histological reports bearing on such complicated cases have little value. (For examples, see Gaillet,[15] Reny,[16] Baillet,[17] Kauffmann.[18])

Limiting the term gynecomastia, then, to hypertrophies of the

[10] Provided, of course, the nullipara had never secreted true milk. For a discussion of milk secretion by virgins, see Gellhorn, loc. cit., footnote 1.

[11] In Schuchardt's Collection.

[12] Beitrag zur hist. Kenntnis der sog. Gynäkomastie, Beiträge zur klin. Chir. (Bruns), 1895, xiv, 179.

[13] Zwei Fälle von Hypertrophie der männlichen Brustdrüse, Inaugural Dissertation, Berlin, 1894.

[14] Einige angeborene Anomalien beobachtet im path. anat. Inst. zu Bukarest, No. 7 in Med. Jahrbücher von der k. k. Gesellschaft, Wien, 1875, p. 261.

[15] Soc. de biol., 1850.

[16] Thèse de Velpeau, Paris, 1880, p. 35.

[17] Bull. de la soc. anat., 1890, p. 532.

[18] Lehrbuch der spes. path. Anat., 1904, p. 955.

male breast, such as may occur as independent anomalies early in life, or as accompaniments of other signs of female habitus, such as feminine voice, female hair distribution, etc., or in association with cryptorchism, hypospadias, hermaphroditism, or other anomaly of the genital tract, or such hypertrophy as may occur at any time as a result of castration or disease which destroys the testicles, we may say that the specimen under consideration is not gynecomastia. It differs in that it was painful and tender from the outset. The mass was sharply circumscribed and dense. Under the microscope the stroma is seen to be rich and active, the epithelium to be in a marked state of proliferation.

These observations were controlled by comparison with a genuine case of gynecomastia which Prof. Chiari kindly lent from his private collection, and also by comparison with a second case of gynecomastia herein described (Case VII). It may be stated here that these cases confirm the findings of Stieda, Israel, and others.

III. ADENOFIBROMA. The density, circumscription, and histological structure tally closely with those of an ordinary adenofibroma, such as may occur in the breast of a young woman. The symptoms and course also offer nothing incompatible with such. The only difficulty is encountered when one attempts to make such a diagnosis fit in with the clinical facts which have been published concerning that affection known as "mastitis adolescentium," which also closely resembles this case, but which is supposedly a self-limited affection, it being alleged that most cases recover spontaneously. Further discussion will follow a short presentation of the facts concerning this latter disease.

IV. ADOLESCENT TUMOR. (Mastitis adolescentium, mastitis pubescentium virilis, Albers.[19] Areolar and mammillary tumor, Cooper.[20]) Hochenegg[21] says: "Mastitis adolescentium constitutes an inflammation of problematical etiology which affects the mammary glands of young men." The observed characteristics of the disease will be evident from the following citations taken mainly from Schuchardt's[22] review of male breast affections.

C. Langer[23] states: "At puberty there very often occurs a dull, easily borne pain in the breast, during which time the organ enlarges somewhat, the nipple projects, and the areola becomes deeply pigmented or even reddened. After two to three weeks all symptoms disappear, but the nipple may remain prominent from that time forth." This is common knowledge. Severer cases may be marked by exaggeration of the pain and prolongation of the course. Albers

[19] Mastitis pubescentium virilis, Med. Korrespondenzbl. Rhein. und Westphäl-Aerzte 1843, No. 13.

[20] Lectures on Surgery, 1836.

[21] Lehrbuch der spes. Chir., 1907, i, 869.

[22] Ueber die Vergrösserungen der männlichen Brüste, Langenbeck's Archiv, 1885, xxxi, 159.

[23] Ueber den Bau und die Entwickelung der Milchdrüse bei beidern Geschlechtern. Denkschrift der Kais. Akad. der Wissens. (math. wissens. Klasse), 1852, iii.

found the gland enlarged in the form of "a flat hard callus" lying behind the nipple and tender on pressure.

A case of Lauenstein's[24] reported by Schuchardt ran as follows: The patient, aged twenty-six years, entered the hospital, October 13, 1884, on account of a tumor in the left breast which had developed within five months. No history of syphilis or traumatism. The tumor had never been painful until about the time he sought medical advice. The region of the left breast bulged somewhat. The centre of the left mamma was occupied by a flat tumor the size of a 5-mark piece which seemed not to be fixed either to the skin or to the pectoralis. No pain on pressure. . . . The patient had in each axilla an enlarged freely movable lymph gland, and on the right side a swollen cubital gland. Extirpation October 14. The tumor included the centre of the gland; it was the size of a 5-mark piece and 1 cm. thick. In substance it was dense like a fibroma and sharply defined from its surroundings. On cut surface it looked grayish white. . . . Examination of sections made after hardening showed excessive amounts of connective tissue ground substance, in which gland tubules were here and there introduced and in which in addition there occurred numerous smooth-walled irregular cavities which gave the whole section the appearance of being perforated like Swiss cheese. The vascular development in the tumor was not very rich. The diagnosis *fibro-adenoma cysticum*, herewith made, was confirmed by Prosector Dr. Fränkel, of Hamburg. The patient had no further trouble. Schuchardt looks on this case as one of adolescent mastitis.

Stümcke states that the disease lasts until adult life is established; that its course will be shorter the less time there remains before that event. He never saw a case that did not get well spontaneously.[25]

Risel[26] also reports a case like Lauenstein's, in which the tumor was much larger but nevertheless disk-shaped, attached to the nipple, otherwise free, sharply circumscribed, dense, etc. Risel says he too has seen other cases of the disease, but never had to operate except in this instance.

Such cases as Lauenstein's and Risel's are undoubtedly instances of the same disease as Case I. They were also looked upon by the writers as identical, for a time at least, with adolescent mastitis. If such cases as these were capable of spontaneous healing like the rest, it would be difficult to think of them as tumors, in spite of their structural resemblance to adenofibroma. But it will be noticed that those cases which were operated upon and showed tumor structure had already demonstrated clinically that they were dif-

[24] In Schuchardt's Collection, loc. cit.

[25] It has also been said that onanists and those who have exercised the sexual function are seldom affected.

[26] In Schuchardt's Collection, loc. cit.

ferent from the ordinary adolescent mastitis, and were operated upon for this very reason, that is, their lack of self-healing tendencies.

Microscopic examinations have never been made in such cases as get well by themselves. If made, it is probable that the picture would be found to differ from that of such cases as Case I. This unknown picture would be that of adolescent mastitis.

Briefly summarized, Case I is an instance of adenofibroma in the male breast. What "adolescent mastitis" is, we do not know. Case I throws no new light on it, unless it be taken to show that "adolescent mastitis" may occasionally give rise to true adenofibroma.

A review of available literature made for the purpose of learning how frequent these cases of adenofibroma are, showed that while malignant tumors occur in the male breast about one-fiftieth as often as in the female breast, and are hence not extremely rare, non-malignant neoplasms (in which we do not include adolescent tumor) occur much less frequently. Pure undoubted adenoma has been reported three times only (Römer[27]), and one of these reports was suggestive of carcinoma. As stated at the beginning of the paper, reports of "adenofibroma" have included many sorts of affections, so that it is difficult to judge of their frequency. Some half-dozen cases in the literature had characteristics which made it evident they were identical with the one in hand. But probably they are commoner than the literature indicates.

Case II.—Museum specimen 3923, dated June 10, 1902. Histological Protocol 1756, Dr. Boeckel.

Clinical History. Edward S., aged ten years, was first examined by Dr. B., June 6, 1902. The mother said that a tumor began in the left mamma eight months before, and that recently it had begun to grow larger (no mention is made of pain). Examination showed a tumor in the upper part of the left mamma the size of a small apple. The nipple protruded. Axillary glands not enlarged. Clinical diagnosis adenoma. Extirpation June 10. Complete healing of wound. No recurrence after four years.

The tissue consists of a roughly oval disk with one surface a little concave, the other convex. Dimensions, 8.5 x 5 x 2 cm. The surfaces are everywhere smooth, in places slightly knobbed. All signs point to a sharp circumscription. The cut surface is marked by connective tissue trabeculæ, which subdivide the whole into small irregular areas in which the tissue is darker and more opaque than the rest, corresponding to ducts and terminals.

Microscopic examination shows trabeculæ of relatively dense connective tissue provided with numerous, fairly plump nuclei, and moderate numbers of bloodvessels (Figs. 3 and 4). Fat tissue occurs in the stroma as long islands running parallel to the course of the fibers and as single fat cells or small clusters of the same. The

[27] Inaugural Dissertation, München, 1880.

glandular parts show the same characteristics as those seen in Case I, except that in this case the acini are better developed, groups of eight, ten, and twelve being not uncommon. These are

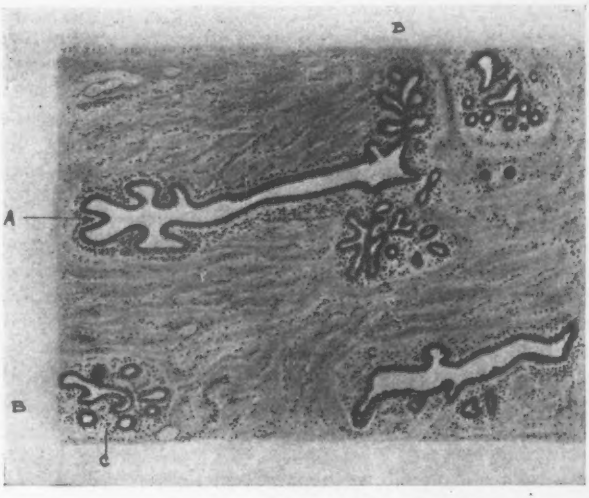

FIG. 3.—Case II. *A*, zone of active proliferation of stroma cells; *B*, group of characteristic end-sacs—virgin type; *C*, mantles of loose tissue containing blue-staining fluid. (Zeiss ocular 2, objective AA.)

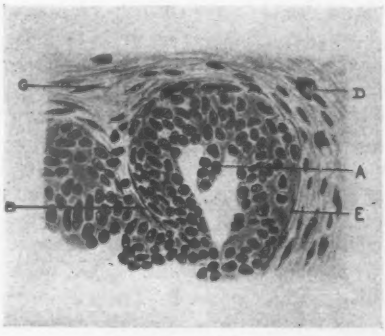

FIG. 4.—Case II. Shows true epithelial proliferation *A*, and papillary invaginations in a duct *B*. *C* shows the character of the blue-staining periglandular mantle. *D*, young connective cells. *E*, basement structure. (Zeiss ocular 4, objective DD.)

larger than those seen in Case I, though not like those of a functionating female breast. The glands are surrounded by the same blue-stained mesh containing numerous young connective-tissue

cells. Elastic fibers are absent. The bloodvessels are full of blood, and many show in their vicinity a slight sprinkling of leukocytes. The gland ducts contain gray detritus and drops of coagulated fluid, which stain deeply with eosin.

This case differs in no essential from the foregoing except for the larger size of the tumor and for the slight round-cell infiltration. The high-grade development of rudimentary acini is no more than has been observed in normal breasts. Diagnosis, Adenofibroma.

CASE III.—Museum specimen 3877, dated June 13, 1899. Label: Adenofibroma mammæ dextræ.

Clinical History. A tumor developed in the breast of a soldier, aged nineteen years, during the course of a year. The patient was examined by Dr. Eichel, who found in place of the right mamma a disk of firm consistence measuring 7 x 7 x 2 cm. It was not well defined from the surrounding tissues. The nipple was normal, the areola pigmented deeply. The axillary glands were slightly enlarged. The tumor was extirpated, the axilla opened, and the enlarged lymph glands removed.

The tissue of the tumor is dense and white. It sends prolongations into the fat tissue, which in turn projects into the tumor. Examination of the cut surface, which is of a translucent gray-white color, reveals irregularly distributed linear, y-, or glove-shaped flecks of yellowish color corresponding to gland structures.

Microscopically fibrous tissue predominates to a greater extent than in either of the foregoing cases. It is of uniform consistence throughout and sparingly beset with thin, narrow nuclei. Bloodvessels are small and few in number. The glandular parts consist mainly of long dilated ducts branched dichotomously. Occasionally they show lateral twigs. End-sacs occur in groups of only two to four. The papillary structures described in Cases I and II are less marked. Fat is scarce, elastic tissue absent. Scatterings of leukocytes occur in several places. The lymph glands show areas of hyperplasia.

In this case the patient's age is the same as in Cases I and II. While there is no specific history of traumatism, it is not at all improbable that such had occurred, since a tightly clothed soldier in the course of drill is likely to rub or bruise the chest. The sharp circumscription of Cases I and II is lacking, nor is the stroma so active nor the epithelial changes so marked. There is, moreover, a diffuse leukocytic infiltration, while the axillary glands examined showed hyperplasic areas.

Another case, with no clinical history available, but, so far as ascertainable, like the last, may be introduced here and discussed along with it.

CASE IV.—Museum specimen 3925. Male, aged eighteen years. Dated, June 11, 1905: labelled, Adenofibroma mammæ. The specimen consists of an ellipse of skin, including a prominent nipple with

a pigmented areola. Beneath the nipple is an ill-defined, tough, whitish mass, which shows under the microscope the same structures as Case III.

Such cases as these (III and IV) differ less markedly from inflammatory processes than do such cases as I and II. Morphologically they stand on the dividing line between inflammation and tumor. They are entirely analogous to the more diffuse adenofibroma-like growths which occur at various times of life in the female breast. The completeness of the series of forms intermediate between inflammation and tumor in the female breast has even been made the basis for the view that direct metamorphosis of fixed tissue cells into tumor cells may occur (Fabian). This view is only quoted to illustrate how many the intermediate forms are, and to point out that in the male breast also we should expect to find a series of tumor-like growths, some showing more, some less, resemblance to pure inflammation, on the one hand, or to adenofibroma, on the other. Such cases as the foregoing are taken as evidence that such a series does exist in males as in the other sex. They throw no new light on the real nature of such.

The four cases so far described occurred in youth, and seem to fall into two groups, two sharply defined tumors, two ill-defined, the former associated with no history of traumatism, the latter perhaps traumatic. The two following cases are of frankly post-traumatic enlargements in adult breasts and seem to resemble Cases III and IV rather than I and II.

Case V.—Museum preparation 3926. Male, aged fifty-six years.

Clinical History. Nine months ago patient fell, striking against the left breast, after which a tumor developed very slowly. Lately it has shown a more rapid growth. October 11, 1898, extirpation by Dr. Lange, of Strassburg, who found the mamma represented by a tumor the size of a hen's egg, not sharply defined from the surrounding tissues. The nipple was prominent and adherent.

The tissue is a dense yellowish mass 4.5 x 2 x 2 cm., which blends intimately with the fat tissue surrounding it. The cut surface shows trabecular markings, the interspaces between which are of darker color and irregular shape.

Microscopic Examination. Dense connective tissue stroma predominates. It is fairly vascular and beset with numerous elongated but not extremely thin nuclei. Gland elements occur as ducts and terminals, as described in Cases I to IV, varying according to the region from which sections come. They are enveloped in broad mantels of loose meshed tissue composed of narrow spindle cells with fibrous extremities, in the interspaces between which, especially near the basement membrane, young connective tissue cells occur. The whole mesh also contains the blue-staining substance described in Case I. Fat tissue is seen in the dense tissues

in moderate amounts, especially near the periphery, where it occurs in large islands. Toward the centre one sees only single fat cells or small groups of them. The glandular parts correspond closely to those of Cases I, II, and IV. Elastic tissue is lacking. A few scatterings of leukocytes occur.

Case VI.—Johann S., aged forty-two years, has noticed a tumor in the left breast since a fall three months ago, in which the breast was bruised. It has grown a little ever since. The patient is otherwise normal, of strong build, and decided masculine habitus. Examination reveals a palm-sized tumor, which is freely movable, on the deep tissues and under the skin. The nipple and areola look large, but are not adherent. May 7, 1907, extirpation. Healing.

The tissue sent to the laboratory consists of an ellipse of skin containing nipple and areola, both of which are prominent but not fixed to the mass which lies below. Under the skin is a piece of tissue 8 cm. wide and 5 cm. thick, in the centre of which is an indurated mass corresponding to the mammary gland. The latter blends all around with the fat tissue. It is firm and consists mainly of fibrous tissue enclosing elongated, semitransparent, colloid-like areas. In the centres of these areas the gland ducts are visible as clefts, or punctate orifices which can be sounded with a bristle. Microscopically, the findings are essentially the same as in Cases III and IV.

In their lack of circumscription these two cases (V and VI) resemble the two preceding cases (III and IV). The only difference between them and Cases III and IV is that the latter occurred earlier in life, while their histories fail to mention the occurrence or non-occurrence of traumatism as an exciting cause, while Cases V and VI arose in advanced life as a result of definite injury. Probably Cases III, IV, V, and VI all belong to the same category, that is, the post-traumatic enlargement just discussed.

These cases differ from adenofibromas like Cases I and II in that the latter began spontaneously as affections which, seen early, would have been called "adolescent mastitis," and developed into sharply defined disk-like tumors.

Consideration of these cases in conjunction with case reports in the literature suggests the view that such sharply defined adenofibromas as Cases I and II always originate in this same way, whereas the ill-defined less certainly newgrowth-like enlargements are frequently a result of traumatism. The sharply defined growths are closely analogous to the painful adenofibromas which occur in young female breasts, which it may be incidentally noted, also frequently begin at about the time of puberty as a result of an affection resembling in all ways "adolescent mastitis." Jopson, Speese, and White have recently noted the relatively great frequency of fibro-epithelial enlargements of the breasts of children at the

time of puberty as against other periods in both sexes. The ill-defined growths are analogous in every way to non-malignant indurations which occur in adult, less often in young, female breasts. Both can be looked upon, however, as adenofibromas because of their characteristic structure and progressive growth.

The last case, an example of gynecomastia, is here recorded because reports are still needed definitely to establish the nature of this rare affection. Stieda's two cases in 1895, with six more collected by him from the literature, form the bulk of what is known concerning the histology of the genuine affection. It also offers favorable opportunity for direct comparison of this process with the other cases of the adenofibroma-like group herein described.

CASE VII.—Museum specimen 3359, from the Pathologic-Anatomical Institute. Labelled Hyperplasia mammæ.

Autopsy protocol, November 17, 1894; male, aged fifty-nine years. Cause of death, cirrhosis of liver; also, hypertrichosis and enlargement of both mammæ. The specimen consists of an ellipse of skin including the areola 2.5 to 3 cm. in diameter, and a prominent nipple 3 mm. thick. Around the edge of the areola is a growth of long, coarse hairs. Under the skin is a mass of fat tissue including a gland 6 x 7 x 1.5 cm. not more sharply defined from the surroundings than a normal gland. Its consistence is much less firm than that of Cases I to VI. More cannot be said, owing to the preserving fluid.

Microscopic sections show even to the naked eye a light loose stroma especially marked in the deeper parts of the gland where the ducts bifurcate, so that the peripheral branches of the glandular elements seem to be supported in a light mesh. Under the microscope the relatively dense stroma directly under the nipple is found to consist of wavy bundles of coarse fibers with scanty numbers of long thin nuclei and few bloodvessels. Deeper down such stroma occurs only in trabecular strands, between which, and hence enclosing the gland-parts proper, are areas of very loose fibrous tissue provided with very few nuclei. The whole is more lightly built than the normal breast of a fifteen-year-old boy, pictured in Fig. 1. The ducts show one to four layers of low columnar or polygonal epithelial cells resting on the basement membrane. There are none of the epithelial prominences described in Cases I to VI, nor any invaginations of the duct walls into the lumens comparable to papillæ. Fat is seen in patches lying parallel to the course of the fiber bundles, much more plentiful peripherally than in the centre of the gland. No leukocytes are found.

This specimen differs from the normal only as regards gross measurements, and the texture of its stroma. The latter may depend on the different ages of the subjects compared. The differences between this case and Cases I to VI are essentially the same as the differences between the normal gland and Cases I to VI. These

findings are virtually in accord with those of Israel, Scheiber, Stieda, and Volkmann. They confirm the view that gynecomastia is simply hypertrophy.

SUMMARY. Seven cases of non-malignant enlargement of the male breast are reported, affording opportunity for a comparative study of the members of a rather rare and little studied group of affections. Six of the cases are adenofibromas as here interpreted, but they admit of division into two groups:

1. Sharply circumscribed, firm, dense tumors, which arise without traumatism in the breasts of young men, run a painful clinical course, and show under the microscope little or no evidence of inflammation, but all the characteristics of ordinary adenofibromas, such as may occur in the breasts of young women, and to which they are here looked upon as analogous. Cases I and II are of this type.

2. Diffuse or ill-defined growths which may occur at any age as a result of traumatism. Microscopically, these enlargements have virtually the same structure as those of Group I. Signs of inflammation are more in evidence, however, so that their differentiation from chronic inflammation may not be easy (Cases III to VI inclusive). They are here called adenofibromas, because of their progressive growth and microscopic structure. This group corresponds to the "traumatic indurations" of some writers.

Cases like I and II are rare. Cases of the type of V and VI are probably more common than is generally supposed. The early stages of cases such as I and II closely correspond to the disease of problematic nature and unrecorded histology known as "adolescent mastitis." Case I in particular with its small disk-like tumors showed all of the characteristics which have been assigned to this affection.

What the relationship is between adolescent mastitis, so-called, and adenofibromas cannot be settled until histological examinations of the tissue in the former disease have been made. Certainly a close relationship exists between adolescent mastitis and tumors such as are included in Group I. Probably adolescent mastitis is a separate affection, as a sequel to which adenofibroma may develop in exceptional instances. Nearly all cases of sharply circumscribed adenofibromas of the male breast heretofore described have developed in youth in the course of what appeared to be "adolescent mastitis." Further study of this affection is needed. Case VII is an example of gynecomastia. Its histological structure is simply that of an hypertrophy which confirms the few exsisting reports on the histology of genuine unmixed examples of this rare condition. The term gynecomastia might well be dropped in favor of hypertrophy.

OCULOMOTOR PARALYSIS ACCOMPANIED BY FACIAL PALSY, NEUROPARALYTIC KERATITIS, AND HEMIPLEGIA.[1]

By Burton Chance, M.D.,
ASSISTANT SURGEON TO WILLS HOSPITAL, PHILADELPHIA.

Through the kindness of Dr. Schwenk I am allowed to report the following case, interesting on account of the multiplicity of important symptoms, the vagaries in the modes of their production, and the marked regain of power by the patient.

On August 24, 1905, a tall negro woman came to the hospital complaining of the sudden closure of her left eye. The eye had been inflamed for six weeks past. Two weeks before coming to the hospital the sight was totally obscured by the falling of the lid over it. The woman was aged thirty years, a housekeeper, and had been married for eight years. She had been pregnant only once, six years ago. Two days before the end of term she fell down stairs and gave birth to a dead child. She denied having specific disease. She had been in good health until March, 1905, when she was seized with violent pains in her head, and was treated for rheumatism. In July her left eye became inflamed, and she had applied for treatment at another hospital; this inflammation was not very painful.

On examination the left brow and cheek were seen to be discolored, and near the outer angle of the orbit there was a large circular maculation, which had resulted from long blistering. The eye was completely covered by the upper lid. There was facial palsy on the left side and the mouth was drawn toward the right. When the lid was raised the globe was seen to be rotated outward. It was deeply injected, and the epithelium of the cornea was macerated and steamy over nearly the entire surface. The summit was œdematous and infiltrated; and it stained deeply when fluorescin solution was instilled. No view of the fundus could be obtained, nor could the iris be seen. Atropine suspended in oil was prescribed, and the patient was instructed to take a saturated solution of potassium iodide, beginning with fifteen drops and rapidly increasing the dose.

The periphery of the cornea was less œdematous in two days, and soon became sparkling. The ulcerous area rapidly became sharply outlined. In a few days, on forced effort, there was slight ability to raise the upper lid, but no effort could overcome the paresis of the internal rectus, though there was a faint reaction of the superior oblique muscle. By September 21 the ball remained divergent and fixed at the outer extremity. Facial paralysis was complete on the left side, and in the distribution over the superior and inferior

[1] Read at a meeting of the Wills Hospital Ophthalmic Society, Philadelphia, March 2, 1909.

maxillary branches of the fifth nerve there was anesthesia. The cornea continued hazy and anesthetic. About this time the patient's left arm and leg became numb and powerless. She was seized suddenly in the night in the midst of a severe headache; in the morning she could not lift her arm nor raise her leg in walking.

On September 28 the patient was sent to Dr. Wm. G. Spiller, at the Polyclinic hospital, for an examination of her general state. His letter, dated October 12, reported that the patient had cerebral syphilis. On October 24 the solution of atropine was discontinued, and by November 11 the "mixed solution" which had been ordered three weeks before was withdrawn, and mercurial ointment substituted. By November 28 the patient showed signs of ptyalism, and all medicines were withheld. Toward the end of December, when efforts were forcibly made the globe could be brought to the vertical line, but not within the meridian, and when the exertions were made it was noticed that the palpebral fissure widened so that the globe was entirely uncovered by the lid. At this time marked increase in the general muscular power began; the facial palsy was reduced, and the leg and arm regained much of their lost functions.

On January 5, 1906, the cornea again became denuded. To allay the irritation the lids were sealed with plaster and a firm bandage was kept on for two days; on the removal of this the exposed area was seen to be closed, but the surface was shaggy, as though it were studded with epithelial shreds like the remains of ruptured herpes. These shreds were examined by Dr. Goldberg and found to consist of epithelial cells only. Toward the end of the month the epithelium became restored. From this time on the return of power was marked. The ptosis disappeared almost entirely. The eye could be rotated to the primary position, but not within the vertical nor above or below the horizontal meridian. The woman could walk with greater comfort. A certain childishness manifested in the past gave place to a decidely acute intelligence. Before the first of February, 1906, the patient ceased her visits, and did not return again until January, 1909, in response to a letter from me. By this time she had regained all her lost weight and much more besides, for she weighed 215 pounds. In the past three years she had been able to keep house and to work as a laundress. She has had a constant dull ache over her left brow, and, when overtired, she has attacks of headache, and the arm and leg feel like "pins and needles." She is very much annoyed by diplopia when she attempts to look up or down, yet even in the primary position there are two images, the left being the false one and the higher. The sight is good, but with the left eye objects appear far away and small. There is now partial ptosis when at rest, but by wrinkling her forehead she can draw the lid up to the limbus. There is paralysis of the superior and inferior recti, the inferior oblique, and the superior oblique muscles; the ocular excursions are limited to the extent of free adduc-

tion and abduction, while supraduction is abolished and infraduction is only faintly perceptible. The diplopia is upward. There is no accommodation power, for while the woman can read types 0.50 D. with the right eye, she has great difficulty in seeing type 2 D. with her left. The vision, however, in each eye equals 5/10. The pupil of the left eye is not circular and is apparently fixed. The fundus is healthy. The cornea is perfectly smooth, and, even with the stereoscopic loupe, nothing is found marring the clearness of the membrane. It is, however, anesthetic, and so is the conjunctiva. There is a free flow of tears. The adjustment of spherical lenses + 1.25 D. with which she sees 5/5, has relieved the woman in the past month of this annoying epiphora. The facial movements have been regained; she can wrinkle her brows and protrude her tongue straight. The face is anesthetic to pin pricks, including the nose, tongue, all of the upper part of the head except that portion comprised in a line from the lobe of the ear and ramus of the jaw forward to about three-fourths of an inch from the meridian line of the chin up beyond the angle of the mouth, thence obliquely to the ear.

In order to know the patient's general condition, I sent her to Dr. T. H. Weisenberg for an examination. He fortunately remembers having seen the woman in Dr. Spiller's clinic at the Polyclinic hospital four years ago. As Dr. Weisenberg points out, the separate occurrence of left hemiplegic with left oculomotor palsy is most unusual. It is probable that the hemiplegia was caused by vascular disturbance in the right side of the brain, while the oculomotor palsy arose through a disturbance at the base on the left side, in advance of the nerve fibers supplying the extremities. The hemiplegia has almost entirely disappeared; it is rather strange, therefore, that the oculomotor disturbance remains. This palsy has been most extensive, affecting both the intrinsic and the extrinsic mechanisms. The widening of the palpebral fissure during the middle period of the ptosis was rather startling in appearance, and was the first instance of that phenomenon in my experinece. The facial palsy has all but disappeared, the anesthesia being the most annoying subjective symptom in this connection. It is most fortunate that the early keratitis did not extend to the true corneal tissue.

INSANITY, RESPONSIBILITY, AND PUNISHMENT FOR CRIME.

By James J. Walsh, M.D., Ph.D., LL.D.,
DEAN AND PROFESSOR OF NERVOUS DISEASES AND OF THE HISTORY OF MEDICINE IN THE FORDHAM UNIVERSITY SCHOOL OF MEDICINE, NEW YORK.

To most people it seems very startling when they are told that experts in mental and nervous diseases are agreed that no absolute definition for insanity can be given. Insanity, which means etymologically ill health, has now come to be limited exclusively to mental ill health. It is very evident that this is a term of extremely wide comprehension. It shades off on all sides into normal conditions so elusively that it is impossible properly to set limits to it. The expert may not quite agree that "half the world is off and the other half not quite on," nor with that expression of the French that "we are all a little cracked, only some of us conceal the crack better than others;" but he does not like to be pinned down to a formula of words according to which the sanity or insanity of individuals may be judged with absolute assurance. As a matter of fact, no definition will stand the light of criticism and discussion and application to all the varying cases that are seen.

The confession that there is no satisfactory definition for sanity or insanity even by acknowledged authorities in mental diseases, loses most of its original startlingness as soon as we are brought face to face with the fact that there is also no definition for health or illness of body. After all, the two problems are very nearly alike. The one is mental and the other physical ill or well being. Sanity of mind cannot be defined, but neither can sanity of body. Here once more there are so many elements that enter into the problem, differing in each individual, that they cannot all be combined in a single formula. This is, after all, nature's way in practically everything. Plants and animals differ from one another ordinarily quite as strikingly as sane and insane individuals, yet no absolute definition by which plants may be distinguished from animals in that borderland where the two kingdoms of living things touch, can be framed. Nature evidently did not intend that there should be such precise delimitations in nature. Things grade so insensibly into one another that it is impossible to make fine accurate distinctions. It is the individual that must be studied and not the class, and the various expressions of nature animate and inanimate have an individuality all their own.

It is evident then when we talk of insanity as a defence for crime we are turning to an excuse which may be extremely difficult to control and keep from being abused. It is often said that an insane person is not a criminal and that he must not be held to criminal responsibility for his actions. While the medical expert will not and

cannot give a definition of insanity, and does not hope ever to be able to give one, he does not hesitate to tell us who are or are not insane. It is evident, however, that in the haziness of the subject it is extremely easy for the expert, even in good faith, to be led when his interest and that of his client, for it is as client rather than as patient that the medical expert's employer must be considered, sway his judgment, that he may be easily led into expressions which can only serve to be a source of confusion to the court and above all to the jury, and not at all a source of such expert knowledge as would prove to be of service. Here is where the difficulty lies in the great abuse of the plea of insanity, which has been allowed to creep into our criminal procedures in recent years.

Between this and the readiness with which lawyers find legal quibbles and technicalities to delay and defeat justice, these two abuses have brought about a very serious state of affairs. The number of murderers who are punished in this country is very small indeed. A prominent divine in New York pointed out not long since that it was much safer to be a murderer in this country than to be an employee of a railroad. When his statement was questioned, he showed by the statistics of recent years that to the whole number of brakemen employed in this country, more were killed every year than of murderers were punished to the number of murders. One of the things that most impressed Ambassador White when he returned to this country was the great number of murders committed and the few murderers that are punished. He called attention to the fact, which is not pleasant for American ears to hear, that human life is cheaper in this country than in any other portion of the civilized world, with the possible exception of Vendetta-ridden Corsica or Sicily. It is evident that without realizing it we have been slipping back into a barbarism none the less to be deplored because it happens to be associated with an over-refinement, which it must not be forgotten has characterized the history of all nations which, having reached an acme of progress, have begun to decay.

Of course, it is realized that many of the murders committed in this country are due to an immigrant population and to the fact that these peoples have not as yet come under the influence of the civilizing process at work in this country. It must not be forgotten, however, that many of our most prominent murders are committed by native-born citizens of good education and with all the advantages that our civilization is supposed to give. Most of these murderers, however, escape on the plea of insanity. We are in the midst of a reaction in this matter. Time was when the rule was an eye for an eye and a tooth for a tooth, and for a human life a human life, as if somehow a crime must be compensated for. We do not care to make the punishments fit the crime now, but the criminal, and we have gone so far to the other extreme that whenever there is anything suspicious in a man's previous history we are prone to think that this may

have impaired his responsibility to such a degree that he does not deserve punishment. It has been easy, therefore, for the medical experts to lead us into a state of mind where criminals escape punishment very readily on the plea of insanity—and then secure their release from insane asylums because they are sane.

There is a tendency to blame physicians for this state of affairs, and to say that is it due to a commercializing of professional dignity. Perhaps there is some truth in this, but it would be entirely unjust to lay the blame at the door of the medical profession alone. The physician is bound by a professional duty to protect the public and secure the ends of justice. There is no doubt of this, but he is not bound as formally to secure the enforcement of law and the ends of justice as is the lawyer. It is clear that at the present moment somehow the legal profession has been diverted from its true purpose to a very large extent. This is so true that ordinarily it is quite startling for the community to be reminded as it was so forcibly by Governor Hughes in his address to the graduates of Fordham University Law School, in the spring of 1908, that the lawyer on his admission to the bar becomes a sworn officer of the court for the securing of justice. He is bound by oath to strive only for what is just for his client, absolutely nothing more. In all matters in which life and death are not in question he is bound by formal oath to see that justice is done, no matter what may be the consequence to his client.

In spite of this, lawyers find ways and means to defeat justice and to delay it, so that President Taft has declared that injustice to the poor is constantly done. If this is done in mere questions of dollars and cents, it is not urprising that physicians upholding the significance of their profession should, for the purpose of saving life, have exaggerated the meaning of their knowledge of crime and responsibility, somewhat perverted our idea of insanity, and so brought about an abuse of legal procedure that has sadly defeated the ends of justice. Professional life has indeed been tainted by commercialization, but the humanitarian motives that guided the tendency to the present abuse of medical knowledge cannot be impugned. The abuse is not as great as that to be found in the legal profession, and its purpose, at least originally, was much higher. There is much need of reform in the matter of the medico-legal expert, but we may as well start with the realization that this is only a part of the reform that is sadly needed in legal procedures in this country, and by no means the most important part.

The question of responsibility for actions, and above all for criminal actions, is not so simple as many people would like to proclaim it. No two men are free to perform an act or not to perform it in quite the same way. Familiar examples are ready to hand: One man finds no difficulty at all in resisting the inclination to take spirituous liquor to excess; another finds it a most difficult feat, often apparently

impossible for him to refrain from indulging to excess almost whenever the opportunity offers, or at least whenever he gets a taste of liquor. This difference between the two men is founded in their very nature. It would be utterly a mistake to praise the one for his abstinence or to blame the other under certain circumstances for his indulgence. Between these two classes there are others quite different individually. Some of them have a slight tendency, and, fearing the worst, do not indulge in it; some of them have a marked tendency which they are able to resist under most circumstances without very much difficulty once they have made up their minds; some are sorely tempted, fall occasionally, yet never become habitual drunkards. For each of these men there is a different responsibility, and so far as they are to be punished a different punishment must be meted out, for it is our effort in the modern time to make the punishment fit the criminal and not the crime.

This same thing holds true for many other forms of crime. Some men readily lose sight of the distinction between mine and thine, and possess themselves of their neighbors' goods almost without realizing that they have done wrong. They are rare, and we have been accustomed to call these people kleptomaniacs. Between these and the man who hesitates to steal, even when starving or for his starving children, there are many degrees of inclination and disinclination toward stealing. The same thing is true to a more noteworthy degree with regard to anger. Anger, the old saw says, is a brief madness. In English we say very frankly that a man who is very angry is mad. In this brief madness he may be led to do things which he would not do at all in his sober senses. Some men easily get into one of these awful fits of anger in which their responsibility is lessened, while others have a calm phlegmatic disposition from which they are scarcely aroused even by the worst forms of abuse or injury, or even physical suffering.

It is evident in all these cases that in order to measure how much of punishment ought to be meted out for acts committed it is more necessary to know the individual than his act. This often becomes an extremely difficult matter, for after the commission of crime every effort is made to make out as little responsibility as possible for the criminal. The easiest way to do this has been to use the insanity plea. As already stated, we have no definition of insanity. It is easy to understand then that there will a disagreement among physicians as to who is or is not insane, and the result is almost sure to create doubt in the minds of the jury that prevents the proper punishment of crime. Now this system is founded on certain wrong principles as regards the administration of justice. While it is difficult to decide with regard to a man's insanity or sanity, it is not difficult to decide with regard to his punishment when the ordinary purposes of punishment are kept well in view.

The old idea of punishment used to be that of revenge. A man

had done a wrong, and what would ordinarily be held a wrong had to be done to him in order that the scales of justice should be maintained level. At the present time we have no such idea at all. Punishment has two main purposes—the prevention of further disturbance of social order by the particular criminal, and the deterrence of others from like acts. If a man takes away the life of another we do not take away his because thus justice will be obtained, but we take it away to prevent him from ever doing anything of the same kind again. A man who has committed murder is more likely to do it again than another. He has committed one breach of social order; we shall prevent him forever from committing another of the same kind. This is the very best deterrent to such crimes that there is. It will be said, of course, that these men could not refrain from doing their acts. It is doubtful, however, whether this contention is true in the great majority of cases, and the proper punishment of such as occur furnishes the best possible motive to help others from the commission of like acts.

This holds true for children at a time when their sense of responsibility for their acts is as yet undeveloped. They can be taught, even very in early life, by properly applied punishment, that need not be severe, that they must not do certain things, and then they will not do them, or at least, will do them much less. This is true not only for perfectly rational childern, but also for those that are to some degree irrational. Punishment is of great importance in the training of children of low grade intelligence, and there is scarcely any child, however wanting it may be in intellect, that cannot be disciplined into conduct that makes it much less bothersome than would ordinarily be the case. This is well known and it is also well known that the attempt to manage such children without punishment would be extremely difficult, not to say impossible. They do not reason about the thing, they are not quite responsible for their acts, but they do connect punishment with what they have done, and are in many cases deterred from doing it again, especially while they realize that authority is near them and that punishment is inevitable. These are the principles on which the adjudication of punishment for crime must be measured. There is nothing else that can be done if society would preserve itself and its members from those who are irresponsible even in minor degrees.

In this matter practical experience is well worth the while. The lower order of creatures, the animals, we do not consider responsible for their acts in the same way as human beings. We know the value, however, of punishment in deterring them. A dog, for instance, by being whipped a few times when he is young, can be taught not to steal things to eat, and taught that there is an inevitable connection between the taking of such things and the infliction of such punishment. I shall not soon forget my first lesson in philosophy from a dear old professor, who, talking of the memory of animals, demon-

strated that they had a memory, from the ordinary experience of mankind with regard to them. "If a cat does something naughty in your room," he said, "you rub its nose in it, and it will not do it again." The cat had no idea that it was doing wrong. According to its way of life it was not doing wrong. It learned, however, from sensory experience that it must not do this sort of thing under special circumstances, and after the lesson has been once thoroughly learned there is no more trouble of this kind.

Individuals who are of less mental stability than normal require, indeed, more careful discipline than average men. The rational may be managed by sweet reasonableness. The defective child must be made to realize that certain actions will surely be followed by painful punishment, though, of course, the main purpose of modern care for such children is to watch over them so diligently as to prevent them getting into mischief. This is after all what we do with the animals, and we realize the necessity for it. Defective human beings approach the animal in their lessened power to resist impulses, and they must be treated in the same way. If we were to save the animals in an excess of tenderness toward them, because we held to the notion either that they did not know any better or else could not resist their impulses, and then permitted them to do things without punishment, we should either have to get rid of animals entirely, or else life would be one continuous readjustment of things to animal ways. Since defectives occur in the general population, it must be realized that far from being less rigid with them in the matter of meting out punishment for things they do that are harmful to others, we must be even more strict with them. Otherwise, we will have to take the bitter consequences of our own foolishness.

It does not make so much difference if the thoroughly rational individual occasionally escapes punishment for something done, but whenever the subrational escapes, he is encouraged to do it again. More than that, the example of his punishment is needed for others. So far as possible, punishment must inevitably follow crime in the world, in order to impress the subrational and deter them from yielding to impulses. Far from being less deserving of punishment in every sense in which a modern penologist cares to inflict punishment, these individuals are more impressed by it, and, above all, need to be more impressed by it. When the subrational know that they can do things without being severely punished for them, they will always abuse that state of affairs. The thoroughly rational man may be depended on to do his duty as a rule without the need of punishment hanging over him. This is not true for the others, and hence the greater increase in crime, and above all in murder, which has made human life cheaper in this than in any other country in the world, as the direct consequence of recent abuses in our penal system.

It has become very clear now that in recent years we have come

to take entirely too lenient a view in these matters, and that many criminals who deserved to be punished, both because in this way they would be prevented from future crime and others deterred by the knowledge of their punishment, have been allowed to escape justice. The tendency is toward too great mercifulness, which spoils the character of the nation, just as leniency to the developing child spoils individual character. Men may very well be insane, in the broad meaning of that term, in the sense that they have done irrational things, but then there is almost no one who has not. The responsibility of most men for a definite action is quite clear in the sense that if they are punished they will not do it again, or will be less likely to do it again, while if they are not punished their escape becomes a suggestion to themselves and to others to repeat such acts. It is for the subrational that we most need to insist on punishment. The cunning of the insane is proverbial, and this extends also to the subrational, and many of these folk realize that their difference from others, their queerness, as their folks call it, is quite enough to make a verdict of insanity in their case assured with the present lax enforcement of law. If the present state of affairs continues in this matter, we are simply allowing ourselves to be led by the nose by these cunning people into the perpetuation of a state of affairs in which they may do what they like because we have become foolishly oversensitive in the matter of inflicting punishment.

On the principle that punishment deters, a man who has killed another man, even under conditions that seriously impaired his responsibility for the act and with evidence of previous lowered mentality, must never again be free to live the ordinary life of men. He must be under surveillance, and should be confined for life in an institution for the criminally insane. For the subrational such a sentence, if known to be inevitable, would usually be more deterrent than even imprisonment in an ordinary prison for life with all the possibilities for freedom which are presented by executive clemency, pardoning boards, and the like. It is absurd to say that a man may have such an attack of mental unsoundness as will lead him to do so serious an act as taking away human life, and then be expected to get over his mental condition so as not to be likely to do the same thing again. Every alienist knows that this is not true. Such acts, when really due to mental instability, occur either in depressed or maniacal conditions and these, as is now well known from statistics very carefully collected, inevitably recur. Society must be protected from such individuals, and this constitutes the most important reason for punishment. It is no longer a personal matter, but a social requirement.

Conclusions. 1. The term insanity is so vague that its use as a plea to enable the criminal to escape punishment is not justifiable in the present state of our knowledge.

2. Responsibility differs in different individuals, but it is never

quite eliminated except in the absolute idiot. For those of lowered mentality, even the animals, punishment has a good effect.

3. Punishment is not revenge, but is meant to deter the individual criminal, and above all to deter others tempted to criminal acts.

4. Punishment is more needed for those of lowered mentality, of whom the expert may well declare that they are insane, than it is for the normal.

5. Subrational individuals with the cunning of the insane will take advantage of our leniency if present conditions are allowed to continue, and we shall have a riot of crime by personal violence.

REVIEWS.

CONSTIPATION AND INTESTINAL OBSTRUCTION (OBSTIPATION). By SAMUEL GOODWIN GANT, M.D., LL.D., Professor of Diseases of the Rectum and Anus in the New York Post-Graduate Medical School. Pp. 559; 250 original illustrations. Philadelphia: W. B. Saunders & Co., 1909.

THE purpose the author had in mind in the preparation of this work is perhaps better expressed at the beginning of the short chapter on the medical treatment of constipation than it is in the preface. He says: "I believe that drugs properly administered accomplish a very great deal toward the relief and cure of *acute* and *spastic* constipation, but, with a few exceptions, consider them inefficient in the curative treatment of chronic *atonic* and *mechanical* constipation. I am of the opinion that chronic costiveness can be cured in the vast majority of cases without the aid of medicines by means of *education, exercise, psychotherapy, dieting, hydrotherapy, massage, mechanical vibration,* and *electricity*—procedures with which I have already dealt. My experience justifies the belief that drugs are helpful in the treatment of this complaint in certain cases when employed in conjunction with the therapeutic measures named, and further, that they are rarely effectual when used alone."

The non-medicinal methods he mentions are taken up seriatim, a chapter or two being devoted to each. The section on the etiology of constipation emphasizes the facts that digestion may be impaired and the act of defecation delayed or prevented through the influence of the mind upon the gastro-intestinal mechanism when disturbed by anger, fright, sorrow, worry, or depression; and also that the act of defecation may be facilitated by concentration of the mind upon this one object, or delayed when it is diverted by reading or in other ways while in the toilet.

In the chapter on psychic (moral) treatment (the title of which—in conjunction with constipation—irresistibly suggests an "Emmanuel movement") the author repeats the above list of mental factors in the production of constipation, and adds: "In exceptional cases sufferers from constipation become so very anxious to have an evacuation that the anxiety, through its influence upon the nerve centres, produces an inhibitory action upon the motor mechanism of the bowel, and in this way delays or prevents a movement. Such

patients should be advised to occupy and divert their mind in some way as the hour for defecation approaches, and to attempt evacuating the bowel at the very first manifestation of the desire." He says, further, that constipation has been cured by hypnotic suggestion, and also by worshipping and praying to idols, religious relics, the Holy Virgin of Lourdes, by wearing chains, kissing precious stones, or by carrying about amulets which have a reputation for healing certain ailments. His discussion of these subjects is interesting, and presents a pleasing variation in the literary treatment of a subject which does not lend itself readily to fancy or romance.

It must not be thought, however, that the book is not a valuable and practical addition to the literature of the subject. Perhaps when it takes in—as it does—a consideration of splanchnoptosis it is going somewhat beyond the promise of its title. But those sections also are well done, and represent the latest views as to causation, symptoms, and treatment of enteroptosis generally. The chapter on massage is admirable. The teachings as to diet and exercise are among the clearest and most practical with which we are familiar. The various methods and appliances for mechanical vibration, for the use of hydrotherapy and of electricity, and other physical therapeutic procedures, are described exhaustively.

The author is obviously an enthusiast in the direction of the drugless management of constipation and of obstipation—which he defines as "a special variety of constipation brought about as the result of some *mechanical* obstruction to the free passage of the feces through the intestines, as, for example, congenital deformities, stricture, pressure by neighboring organs, tumors, foreign bodies, hypertrophy of the rectal valves, angulation, etc." But his enthusiasm has not made him unreasonable or impractical, and the book may be warmly commended as likely to be of great use to both the surgeon and the general practitioner. J. W. W.

AMERICAN PRACTICE OF SURGERY. Edited by JOSEPH D. BRYANT, M.D., LL.D., and ALBERT H. BUCK, M.D., of New York City. Vol. V; pp. 965, with 452 illustrations. New York: William Wood & Co., 1908.

IN the fifth volume of the *American Practice of Surgery*, regional surgery, comprising Part XVI of the entire work, is commenced. Surgical affections and wounds of the head, by Edward Archibald, M.D., of Montreal, is the opening monograph, and occupies 375 pages; the surgery of the cranial nerves is discussed in 37 pages by Charles H. Frazier, M.D., of Philadelphia; Dr. Charles B. G. de Nancrède covers the subject of surgical diseases, certain abnormities,

and wounds of the face, in a satisfactory and as entertaining a manner as the subject permits; there follow articles on harelip and cleft palate, by James S. Stone, M.D., of Boston; on surgical diseases and wounds of the eye, by George C. Harlan, M.D., of Philadelphia; on surgical diseases and wounds of the ear, by Robert Lewis, Jr., M.D., and on sinus thrombosis of otitic origin, and suppurative disease of the labyrinth, by John D. Richards, M.D., both of New York City; pyogenic diseases of the brain of otitic origin, by Henry Ottridge Reik, M.D., of Baltimore; surgical diseases and wounds of the pharynx, by Charles H. Knight, M.D.; surgical diseases and wounds of the larynx and trachea, by James E. Newcomb, M.D., and on laryngectomy, by Frank Hartley, M.D., all of New York City.

Dr. Archibald has given us, in his monograph on the surgery of the head, an article encyclopedic in character and well illustrated by statistics from the Royal Victoria Hospital, including autopsy and operative investigations of much value. He gives an excellent historical and critical account of the mechanism of skull fractures; and traces our knowledge of the pathogenesis of compression of the brain up to Cushing's work, through the previous observations of von Bergmann, von Schultén, and Leonard Hill. He lays stress on the value of blood pressure in diagnosis and prognosis in cases of compression, and on the fact that the "vagus pulse" is no indication of the *degree* of compression present, merely testifying to its existence; he insists on the value of the depth and rhythm of the respirations as an aid in diagnosis of the degree of compression; and on the fact that though failure of the respiratory centre usually precedes failure of heart action, yet that the former is always due to anemia of the vasomotor centre. The subject of concussion of the brain remains in a much more obscure state than that in which compression has been put by modern investigation, the main symptoms being unconsciousness, with a moderate or rapid pulse, slow and later normal respiration, and low blood pressure. Archibald holds that the immediate unconsciousness is due to anemia of the brain, and that prolonged unconsciousness is caused by a prolonged anemia due to the marked depression of the vasomotor tone, and to capillary thromboses and cerebral (intracortical) œdema; he contends that these symptoms cannot be due to direct injury of the ganglionar cells, since in such case the symptoms would be permanent; he concludes, finally, that the lesions commonly classed as those of contusion are, if present in cases of concussion, mere by-products, not causes themselves of the symptoms of concussion, because they are present also when no symptoms of concussion exist. Should the symptoms of compression appear after those of concussion subside, he urges decompressive craniectomy even in the absence of all focal signs. This is a matter about which surgeons are not agreed; and no doubt more harm than good would be done were the average

surgeon to adopt decompression as a routine treatment. We say the average surgeon, not doubting his mere operative skill, but questioning his ability or willingness to study his cases of head injury so carefully as to enable him to reach a rational conclusion as to which patients he might reasonably hope to save by operation; and we think Archibald does well to insist on the propriety of the surgeon himself exhaustively examining and attentively observing for prolonged intervals patients who are too often left to the entire care of an interne or a nurse, because thought on superficial examination to be hopeless cases. Were surgeons to investigate the mortality of their unoperated as well as of their operative cases of severe head injury, they would be surprised at the mortality, and would be more ready in the future to study in person even apparently doomed cases with a view to their operative relief. These remarks apply as well to fractures as to compression without fracture, and to meningeal hemorrhages due to trauma.

Archibald considers intradural much more frequent than extradural hemorrhages, the former including subdural or extrapial hemorrhages. In discussing the subject of pulsating exophthalmos no notice is taken of the work of de Schweinitz and Holloway (1907), in which they came to the conclusion that ligation of the orbital veins is the best treatment. Archibald still recommends ligation of the internal carotid. He concludes his article with an excellent account of brain tumors, and the technique of cerebral operations.

Frazier's contribution to the surgery of the cranial nerves covers the subject in a satisfactory manner, but the illustrations of excision of the Gasserian ganglion are rather disappointing, making the ganglion appear altogether too easy of access and too near the surface. He gives the mortality of the modern operation as only 3.7 per cent. It is a pity that no bibliography accompanies the text; most of the articles are so supplemented.

Stone recommends that operation for harelip be done between the age of six weeks and three months, and that for cleft palate between six and ten or fifteen months.

The inclusion in a modern system of surgery of articles on the so-called specialties (eye, ear, etc.) is often adversely criticised, but we think the editors are quite justified in not excluding these subjects. This system of surgery doubtless reaches many who have neither modern text-books on these specialties nor specialists at hand to whom they can refer their patients on short notice; and the authors of these sections, with few exceptions, have been careful to confine themselves to such affections as any general surgeon may be called upon to treat. The longest article, that by Harlan, on the eye, is quite an encyclopedia in itself, and, in addition to epitomizing the literature of the subject, is enriched both pictorially and textually from the author's own ample experience. Lewis warns the surgeon in undertaking a "mastoid operation" always to be prepared to open

the lateral sinus, to excise the jugular vein, or to open the skull and explore for cerebral or cerebellar abscess. That this is good advice is not to be heedlessly denied; but it is quite apparent to the general surgeon that the otologist sometimes pushes his operative intervention too far in the case of patients acutely ill; and that better ultimate results will be obtained if at the first operation the mastoid cells and antrum are merely drained, and if the complete operation be postponed until more strength is gained. A. P. C. A.

ARTERIOSCLEROSIS: ETIOLOGY, PATHOLOGY, DIAGNOSIS, PROGNOSIS, PROPHYLAXIS, AND TREATMENT. By LOUIS M. WARFIELD, A.B., M.D., Instructor in Medicine in the Washington University Medical Department, St. Louis, Mo. With an introduction by W. S. THAYER, M.D., Professor of Clinical Medicine in the Johns Hopkins University, Baltimore. Pp. 165; 8 illustrations. St. Louis, Mo.: C. V. Mosby Medical Book Company, 1908.

MONOGRAPH writing has been sadly neglected by the American physicians, yet books dealing with a single subject may be of the utmost value. An essential requisite, however, is that the book should be authoritative. This does not necessarily mean that a name distinguished in medical literature should be upon the title page, but that the writer display a mastery of his subject, not merely the result of reading, but also of personal familiarity.

The present volume does not meet this requirement. It is largely a compilation, and yet only occasionally is the source mentioned. The arrangement is not good. The author does not seem to have understood clearly in some places just what part of the subject he is discussing. Thus, in the chapter on etiology, his paragraph on the congenital form is a mere statement of its existence. The symptoms and physical signs are sadly jumbled, and in different parts of the chapters devoted to them, indeed, in different parts of the book, there is much repetition. It is true that the symptoms and signs of arteriosclerosis are at best somewhat indefinite, but this renders more necessary a clear presentation of the facilities and limitations of our present methods of diagnosis. An unfortunate feature is a tendency to make a statement, and then to retract or qualify it somewhat farther along, leaving the reader in a confused state. The treatment of hypertension throughout the book illustrates this defect.

In the section on prophylaxis, there are a good many familiar platitudes: (page 120) "The struggle for existence is keen," etc.; (page 121) "We live rapidly, burning the candle at both ends," etc. The author lays great stress upon the importance of early diagnosis

and is an enthusiastic advocate of golf in the treatment of the early stage of the disease. He takes a rational view of some of the smaller vices, such as tobacco and alcohol, but believes strongly in the deleterious effects of butcher's meat, and seems to prefer a low proteid diet. Among the drugs, he speaks with approval of two, the iodides and nitrites. To these he adds morphine to meet certain severe indications and an occasional stimulant for heart failure. In the final chapter, entitled "Practical Suggestions," there is considerable preaching. "It is a wise maxim never to drive a horse too far. Apply this to a human being, and the rule holds equally well," and much more of the same sort. The book could have been compressed into less than 165 pages, and yet all the information it contains be well presented. It could have been expanded to a great many more pages with advantage by a more thorough treatment of its subject. The introduction by Dr. Thayer is an interesting piece of writing. J. S.

PARCIMONY IN NUTRITION By SIR JAMES CRICHTON-BROWNE, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy, London. Pp. 111. London and New York: Funk & Wagnalls Company, 1909.

To an American accustomed to the orthography taught in his native schools, the first word as spelled by Sir James in the title of his book appears rather startling; to an American taught to regard Professor Chittenden's researches in metabolism, as he is wont to regard Cæsar's wife, above suspicion and reproach, the contents of Sir James' little monograph appear more startling.

Parcimony in Nutrition is a revised and expanded version of the author's presidential address to the section of preventive medicine at the meeting of the Royal Institute of Public Health, at Buxton, England, in July, 1908. As the title suggests, the book is more or less of a satirical invective against the present-day theory that man has for years been eating in excess of all natural requirements, as elaborated by Chittenden. We hardly turn the second page before the inspirer of the address and its present elaboration can be plainly seen between the lines. But ere our noted physiologist is openly assailed, the avenue of attack lies over the body of an American gentleman, who has "literally chewed himself back into health after being middle aged, obese, dyspeptic, in failing health, rejected by an insurance company, and unaided by many medical men consulted." Sir James with cruel pen and biting sarcasm slays Horace Fletcher in the first chapter, and apart from sporadic resurrections of his victim devotes the remainder of his book to the combat with Chittenden.

It is impossible to review *Parcimony in Nutrition* without a shade of levity. Not that the book is *not* serious, for we believe the author is sincere in his statements, but wittingly or not, he has infused an informal, chatty atmosphere into his work which instructs while it amuses, informs us while we smile. Certain of Chittenden's deductions, arrived at from his painstaking studies, are swept aside with a swish of the pen, and mere statements, unproved by research, are offered in their place. On the other hand, statistics and argument are presented which, it must be confessed, seem difficult to refute. The author points out that Chittenden's contention that the Japanese subsist on a low protein diet is wrong, and he shows that when body weight and body surface are considered there is not much difference between the individual food supply of the Japanese and Voit's standard. The Greeks he shows later on to have been large eaters of meat, despite which fact the group of statesmen, artists, and literary men, who were born 530 to 430 B.C., has never been equalled in any race.

Statistics are not lacking: Chapter III is devoted to the subject of prison experiences (collected from authorities not personal), which show what abundant rations are supplied the convicts; and it concludes with the epigram, "Nothing is more demoralizing than chronic hunger." The author quotes a disciple of Fletcher as saying the British navvy is of little use after forty, and questions, pertinently, "If excesses in diet are this slow in exacting their penalties, may it not be that a subliminal diet may be also tardy in manifesting its untoward effect? May not imperceptible undermining be going on for a long time before there is any obvious collapse?" He places all Chittenden's scientific discoveries against empirical observation. and states that "farmers have valued and cultivated leguminous crops, the four-course rotation of turnip, barley, clover, wheat having been popular two thousand years ago, but it is not twenty years since Hellriegel and Wilfarth revealed to us that leguminous plants bear on their roots nodosities abounding in bacteria capable of fixing atmospheric nitrogen and thus supplying the nitrates essential to grain crops. From the dawn of history men have been engaged in adapting domestic animals to their uses, by making crosses between varieties with differentiating characteristics, . . . but it was only in 1866 that Mendel formulated his law of inheritance in hybrid varieties."

There is an abundance of similar arguments in the book, with a corresponding lack of scientific experimentation to prove the author's point, which is that practical experience and physiological requirements of mankind in general more than offset all the protocols of metabolic studies. The book is well worth the perusal of those desiring to be amused and instructed by this opponent of parcimony in nutrition.

E. H. G.

A Text-book of Surgical Anatomy. By William Francis Campbell, M.D., Professor of Anatomy in the Long Island College Hospital, New York. Pp. 675; 319 original illustrations. Philadelphia and London: W. B. Saunders Co., 1908.

Writers on surgical anatomy tread on dangerous ground; their path is narrow and strewn with stones; on one side lies the barren field of systematic anatomy, from which even the stones have been removed, and on the other is the flowery mead of surgery; and the temptation to step aside momentarily, or even to walk persistently on one side or other of the path usually is too strong to be overcome. The hard task set for himself by a writer on applied anatomy is not only to keep in the path, but to clear it of the obstructions which pure anatomists have placed there, perhaps unintentionally, by their failure to explain the why and the wherefore of the construction of the human body. The applied anatomist attempts in a very real sense "to justify the ways of God to man."

This volume is divided into six parts, treating of the head and neck, the thorax, the upper extremity, the abdomen and pelvis, the spine, and the lower extremity. In general, each subject is discussed under the headings of (*a*) surface anatomy, (*b*) regional anatomy (*e.g.*, shoulder arm, elbow, forearm, etc.); and under each region discussed are to be found short paragraphs on surgical anatomy; but Dr. Campbell pays almost too much attention to pure anatomy, and when he does venture into the fields of surgery it is as one seemingly unfamiliar with many of the practical details of which he writes. This is evidenced, for instance, by his unconquerable habit of making hard and fast rules of surgical procedure based on theories unsupported by clinical experience. Thus, he advises the retention of a plaster cast for several weeks after the reduction of a congenital dislocation of the hip; many months is the usual time. He utters the edict that tuberculous disease of the tarsus requires amputation, and that resections must be confined to traumatic affections; whereas it is well known that recovery is frequent after conservative operations for tuberculosis of the tarsal bones. In doing a tenotomy of the tendo Achillis, he warns against injury of the posterior tibial vessels and nerve; we wonder that he does not caution the student to beware of the external iliac artery in operating for appendicitis! He classifies every tuberculous abscess of the tibia as a "Brodie's abscess."

That the author is not thoroughly familiar with modern surgical literature is shown by his describing gastro-enterostomy as a "drainage" operation, in which the "vicious circle" may be prevented by making the anastomosis at the *most dependent portion* of the stomach; and by his quoting, as if of recent utterance, remarks in approval of Jonnesco's operation for exophthalmic goitre.

In discussing the surgical anatomy of the extremities, Dr. Camp-

bell has done better: his accounts of dislocations and fractures are usually sufficient, if not illuminating; some attention has been paid to the lymphatics; the descriptions of surface anatomy and landmarks are clear; and the illustrations show what is intended. Although all the illustrations are called "original" on the title page, the majority are so only in so far as they have been redrawn from other works. They are all painfully diagrammatic; a few are truly original; and they are the only original things in the entire volume.

A. P. C. A.

GONORRHŒA IN WOMEN. By PALMER FINDLEY, M.D., Professor of Gynecology in the College of Medicine of the University of Nebraska. Pp. 112. St. Louis: C. V. Mosby Medical Book and Publishing Co., 1908.

THIS monograph, dedicated to Neisser, has been prepared by the author to stimulate professional interest in a very important and much neglected subject. He has appended an extensive bibliography, which forms a very valuable feature of the work. It seems remarkable that some such book has not appeared before, since the importance of the subject and the very vague ideas held by the great mass of the profession with regard to many of the problems of sanitation, prophylaxis, and treatment have certainly demanded it. However, the book has at last appeared, and the fact of its publication gives us great satisfaction, since there is no subject in the domain of present-day medicine which needs more careful elucidation. The author offers as an introduction a short but sufficiently complete historical account of gonorrhœa, the next two chapters being concerned with its etiology and pathogenesis; an interesting section upon the pathology is followed by a ten-page description of the course of gonorrheal infection, while chapters upon its diagnosis, frequency, sociological bearings, and treatment follow. The book concludes with a three-page account of the systemic gonorrheal infection and the bibliography before mentioned. In the section devoted to treatment the author has included the conclusions of Butler and Long, based upon their experience with the vaccine method of treatment in the vulvo-vaginitis in childhood. A considerable portion of this monograph is composed of excerpts from the writings of various authors. In many instances they have been quoted verbatim. This occasions some repetition and renders the book a little less readable than would have been the case had the author incorporated the substance of their work alone. While to the specialist there is nothing new in the book, except possibly the vaccine method of treatment, and while certain advice relative to intra-uterine treatment may well be objected to, there is, on the other hand, a very great deal which to the general

practitioner will come as new thought and which will well repay his careful digestion. We feel that Dr. Findley has done a good work in the preparation of this book, and hope that its circulation will be widespread. W. R. N.

SUGGESTIVE THERAPEUTICS, APPLIED HYPNOTISM, PSYCHIC SCIENCE. By HENRY S. MUNRO, M.D. Second edition, pp. 360. St. Louis, Mo.: C. V. Mosby, 1908.

IN his preface the author states that he not only brings to the consideration of the medical profession the results of his personal experience and clinical evidence as proof of the value of suggestive therapeutics in the general practice of medicine, but also a detailed explanation of how to apply suggestion efficaciously, both with and without hypnotism as a therapeutic adjunct. He also states, with a view of forestalling criticism, that his book is not intended principally or even mainly for neurologists or psychotherapeutists, but for the vast mass of the profession to whom this entire field is new.

To criticise this book or even adequately to review it is beyond the ability of the reviewer, who is merely a neurologist. For while it is acknowledged that there is room for an adequate presentation of such a subject one's mature judgment is immediately put in abeyance, and in spite of whatever self command one has, it is almost impossible to read such a book without becoming incensed at such a preposterous presentation. There are so many books which are written nowadays with the presumption that the poor medical man who is asked to buy and read the book knows absolutely nothing. The author, evidently, is one of those who not only thinks so, but actually proves it, for in his text, the better to emphasize to his reader what he wants him to infer, for he presumes that the reader does not use his intelligence, he uses heavy type. As an example of his method we may quote a paragraph taken at random on page 36: "Tell him to relax. Sit down. Sleep on. Doctor, suggest to your subject that when you rub this medicine on his arm three times it will be dead and have no feeling in it as long as he is in this room. Tell him that when he is awake you can thrust a pin in the skin of that arm while he is looking at it, and he will have no feeling in it, that his arm will be perfectly dead as long as he is in this room."

It is a matter of regret that the cure of patients by hypnotism, the use of which the author takes as a matter of course, as if everybody employed it dozens of times a day without the slightest ill effects now or hereafter, in the experience of most neurologists, is not so easy as the author seems to assume. The conclusion that one arrives at after having somewhat painfully perused this book,

is that Dr. Munro, after all, is the only person who really does know anything at all of suggestive therapeutics, applied hypnotism, and psychic science. It is a pity that he did not consider the vexing question of the difference between Christian Science and the so-called Emanuel movement, to say nothing of thought healers, mechanoneurotherapists, osteopaths, and the latest cults of soul massage. And yet this book is now in the second edition!

T. H. W.

THE MUSCLES OF THE EYE. By LUCIEN HOWE, M.A., M.D. Professor of Ophthalmology in the University of Buffalo, New York. Vol. II, Pathology and Treatment, pp. 481; illustrated. New York and London: G. P. Putnam's Sons, The Knickerbocker Press, 1908.

THE second volume of Howe's exhaustive work deals with imbalance of the intra-ocular muscles, extra-ocular muscles (heterophoria), strabismus, paralysis, and atypical movements (nystagmus, etc.), and inflammations and injuries of the muscles themselves. The concluding portion is devoted to operations. An extensive bibliography is added, and pictures and short biographical summaries of one-half dozen eminent ophthalmologists are appended.

The book is not easy reading, but this is not the fault of the writer; but because a degree of preliminary knowledge such as not every oculist possesses is necessary. Careful study of the first volume will supply such deficit to a certain extent, but the questions considered in this volume are more difficult, involving as they do the same problems of anatomy and physiology plus the action of disease and anomaly, much of which is novel and unknown.

Haphazard and rule of thumb methods find no favor with Dr. Howe, and he is entirely correct in the statement that for a proper recognition and appreciation of the importance of the subject of heterophoria, thorough anatomical and physiological knowledge obtained from actual dissections and experiment, the proper appliances, an open mind, with time and patience, are prerequisites.

In view of the exaggerations that have been exploited in some quarters regarding this matter we note with pleasure the sanity of Howe's views. He is not in favor of actively treating every form of muscular deviation which can be recognized, and he admits the limitation of all the means at our command for correcting some of them. The oculist is also admonished not to be oblivious to the effect of general conditions upon the muscles of the eye as well as the reverse.

These two volumes will no doubt find a place upon the shelves of every physician who engages in this kind of special work. There

is room for differences of opinion, but the views expressed in the work should be carefully pondered by all who attempt to treat the anomalies of the ocular muscles. It is much better to pass these by entirely than to attempt their correction in a hit or miss fashion without adequate knowledge of all the factors of the problem. T. B. S.

HANDBOOK FOR ATTENDANTS ON THE INSANE. By the MEDICO-PSYCHOLOGICAL ASSOCIATION, Fifth edition; pp. 390; 19 illustrations. Chicago: W. T. Keener & Co., 1907.

THE English Medico-Psychological Association has filled a distinct need by taking into its own hands the publication of a handbook for the use of attendants on the insane. This is the fifth revised edition. It was not until the second edition that the association took direct charge, intrusting it to a committee of eleven members. The scope of the book has been enlarged in each edition, since the training already given in different institutions has created a demand for some advanced teaching which this book attempts to fill. Its contents are well arranged and include the ordinary instruction in anatomy and physiology of general and nervous diseases and care of the sick. It is needless to review a small book of this kind any more than to say that it is well done and highly to be commended.

T. H. W.

ACTION DE LA CONTRACTION UTÉRINE SUR L'ŒUF HUMAIN, PHÉNOMENÈS PASSIFS DE LA GROSSESSE ET DU TRAVAIL. By DR. PAUL BOUQUET, Médicin de la Maternité de Brest, Directeur du Cours Départmental d'Accouchement. Paris: G. Steinheil, 1908.

THIS monograph covers a subject of considerable theoretical and practical interest to obstetricians. It is written in a discursive style, and its practical conclusions are not such as to add greatly to our therapeutic resources. It should, however, find place in obstetric libraries, and may be consulted to advantage by those studying and writing upon obstetrics.

Bouquet's idea has been to study by references in literature the results of uterine pressure upon the human ovum in its various stages of development. He endeavors to ascertain the effect of the pressure upon the lower pole of the ovum, and considers the results of such pressure in a low insertion of the placenta and in placenta prævia. He further considers the results of painless and painful uterine contractions upon the ovum at different stages of its develop-

ment until birth begins. The phenomena of engagement and dilatation receive considerable attention. The point brought out most strongly by the monograph is the fact that before the rupture of the membranes the fœtus and its envelopes constitute a body of little elasticity; uterine pressure is then exerted without much counter-pressure and becomes essentially a mechanical phenomenon. This observation Bouquet applies to the various phenomena of dilatation and labor.

The practical deduction from his observation is to emphasize the protection given to the fœtus by the membranes and amniotic liquid, and to call renewed attention to the fact that before the membranes rupture resistance of the uterine contents must influence the blood tension of the mother. The author quotes extensively from Varnier, Tarnier, Mauriceau, and others. He offers no original experiments or detailed clinical observation of his own. E. P. D.

MANUAL OF THE COURSE OF STUDY AT THE BANCROFT TRAINING SCHOOL FOR MENTALLY SUBNORMAL CHILDREN, HADDONFIELD, N. J. Arranged by MARGARET BANCROFT and E. A. FARRINGTON, B.S., M.D. Pp. 120. Philadelphia: Ware Brothers Company, 1909.

WITHIN recent years much attention has been paid to the training of subnormal children, and as a result many institutions have been created for this purpose. Of these, the Bancroft Training School is one of the best. The manual under review has been arranged by Margaret Bancroft and Dr. E. A. Farrington, and encompasses the details of instruction used in that school. There is nothing original about it. It is, however, well worked out and shows careful preparation. T. H. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

REGIUS PROFESSOR OF MEDICINE, OXFORD UNIVERSITY, ENGLAND,

AND

W. S. THAYER, M.D.,

PROFESSOR OF CLINICAL MEDICINE, JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND.

A Simple Procedure for the Recognition of Blood in the Urine.—ALBARRAN and HEITZ-BOYER (*Presse méd.*, 1909, xl, 361) point out the need of an easy method for the recognition of blood in the urine, in addition to the test for albumin and the microscopic examination of the centrifugalized sediment. To give the proper interpretation to albumin in the urine the absence of blood must be definitely determined. The microscopic detection of red blood corpuscles is the best, but in vain in case of complete hemolysis. The reagent used is an alkaline solution of phenolpthalein, prepared by boiling phenolpthalein, 2 grams; anhydrous potassium, 20 grams; powdered zinc, 10 grams, in distilled water, 100 grams. The clear, colorless filtrate will keep for months. One c.c. of this reagent is shaken with 2 c.c. of the urine to be tested and 3 or 4 drops of ordinary oxygenated water is added. A red fuchsin color is positive, the intensity depending upon the amount of blood present. It is positive for blood in the dilution of 1 to 100,000. It is specific for blood or hemoglobin, and obtained as well in alkaline as in acid urines. Albumin, pus, sugar, bile pigments, chloroform, or thymol do not affect the test. Uric acid, urates, acetone, indican have no influence, nor do such drugs as the iodides, bromides, salicylic acid, morphine, phenacetin, or urotropin. In a word, of easy execution, specific and delicate, this reaction is an excellent clinical procedure for the recognition of blood in the urine.

Alcoholic Epilepsy.—REDLICH (*Epilepsia*, 1909, i, 41) states that there are certain cases of alcoholic epilepsy in which the abuse of spirits is the only excitant in a person disposed thereto, either through heredity or by a "trauma." In other cases, the use of alcohol may bring about epilepsy

without direct, natural predisposition, either in the form of true alcoholic epilepsy (Bratz) fits appearing under influence of intoxication, or in the form of genuine, constitutional epilepsy, whereby fits also appear, irrespective of spirits being taken or not. Moreover, there are intermediate forms of these types. Redlich discusses especially alcoholic epilepsy. Here the epileptic attack may appear at the moment of maximal intoxication. Emotion and trauma of the head will heighten the action of alcohol. In some cases the fit occurs only during the after-intoxication period, twelve to twenty-four hours after the last dose of spirits. In common epilepsy the attacks very frequently appear at that time. Delirium tremens may simultaneously occur and either this or epilepsy follow on an alcoholic bout. These two conditions are closely related both in cause and nautre. It is a question whether abstention from alcohol may cause fits or not.

Purpura Hæmorrhagica and Hereditary Syphilis.—SABRAZES and DUPERIE (*Arch. d. mal. d. cœur, d. vaisseaux, et du sang*, 1909, v, 257) report the case of a subicteric infant, with fissure at the corners of the mouth, who succumbed to œdema and hemorrhagic effusions. Eight hours after birth the liver, spleen, adrenals, lungs, and skin presented multiple effusions of blood, old and new. They contained spirochetes to the exclusion of other organisms. The multiple hemorrhages are due to rupture of distended capillaries resulting from a mechanical obstruction by the interstitial granulomatous lesions of the organism. The injury done by this septicemia of spirochetes was evident in other organs. The thymus was sclerotic; the kidney glomeruli, and the panceras showed signs of irritation; the myocardium and vasa vasorum of the great vessels contained spirochetes. The relation of hemorrhages to hereditary syphilis has been observed by Levaditi and others, but Sabrazés and Dupérié feel that this case explains more clearly the mechanical origin of the œdemas and hemorrhages. In all these cases the ubiquitous presence of the spirochete is remarkable. If the infant survives, there will remain in the organs traces more or less profound of the multiple lesions —scleroses, defective vessels, collateral circulations tending to hemorrhages, hypoplasia of parenchymas. The infinite variety reveals the vast field of functional insufficiencies, isolated or combined, which may have as origin an hereditary syphilis recognized or overlooked. Rational treatment of the parents and most active and precocious therapy of the newborn are the salvation.

The Small Heart of Cirrhosis.—In three cases of pure alcoholic cirrhosis of the liver, without tuberculosis or arteriosclerosis, CARNOT (*Progrès méd.*, 1909, v, 61) has found at autopsy the heart diminished in size and weighing, respectively, 125, 150, and 183 grams instead of the average 250 grams. In the last case the wall of the left ventricle measured 10.5 mm. instead of 12 mm., and the circumference of the aorta 57 mm. instead of about 65 mm. The small size is readily made out by percussion *intra vitam*. The existence of renal or arterial disease is the exception; when present, causing hypertension and cardiac hypertrophy. The small size of the heart must be in relation to the hypotension in the arteries as pointed out by Gilbert and Garnier. Both are dependent upon the hepatic obstruction which causes a portal hypertension and a

diminution in the volume of blood coursing through the heart in a unit of time. Ludwig, Garnier, and Gilbert have noted a fall in the carotid pressure immediately after ligation of the vena porta. This hypotension lessens the work of the heart, and the diminished size is its adaptation to the lessened function.

Oatmeal in the Curative Treatment of Diabetes.—Von Noorden (*Medical Press*, 1909, lxxxvii, 446) explains the method of procedure in the oatmeal cure, emphasizing the necessity of certain definite rules and the fact that only a certain number of patients are benefited. In 28 per cent. of 400 cases he obtained excellent results. These were cases of medium severity. The treatment does not cure the diabetes, but considerably diminishes its dangers. It is of the greatest importance that from time to time the patients should assimilate large quantities of carbohydrates. Thereby the danger of acid poisoning is averted. For this reason the treatment should be repeated four or five times a year. It requires only a week, as a rule, three oatmeal days, and two vegetable days. Experimental glycosuria was produced in dogs by injecting adrenalin. If a small amount of extract of oats were added, no glycosuria resulted. As there is no doubt that adrenalin diminishes the internal secretion of the pancreatic gland, the probability arises that the oat extract has the opposite effect, *i. e.*, that the extract is a stimulus for the pancreatic secretion. After extirpation of the pancreas in animals, the oatmeal treatment has a very bad effect, and the oat extract does not diminish the glycosuria. This also seems to prove that the oat extract only acts by influencing the internal pancreatic secretion in a direct and specific manner. Therefore, von Noorden presumes that the astonishing effect of the oatmeal treatment is due to the circumstance that with the oats is introduced into the body small amounts of a substance which acts as a stimulant upon the internal secretion of the pancreatic gland.

A Note on the Absorption of Fat.—Whitehead (*Amer. Jour. Phys.*, 1909, xxiv, 294) undertook to determine if emulsified fat was absorbed as such by the intestinal mucosa. The work of Kastle and Loevenhart, demonstrating the universal presence of lipase in the tissues and its reversibility of action to synthesize as well as split fat, has strengthened the view that fat is absorbed in the form of soluble soaps. Decomposed in the intestine it is built up again in the villi. On the other hand, there are no *a priori* reasons why fine emulsions may not be absorbed, and it is well known that the intestinal contents are such as to favor the formation of emulsions. In view of Loevenhart's work, the finding of fat droplets in the epithelium and lacteals of villi does not prove that the fat was absorbed as such. Whitehead fed a cat with butter stained in Sudan III, killed the animal, and sectioned the small intestine. There was an entire absence of red globules of fat in the villi, although masses of red butter still unsplit were evident in the lumen of the intestine. Either the fat had not been absorbed as such or not at all. But by staining these same sections in Sudan III fat was demonstrated in great abundance in the epithelium and lacteals of the villi, in the form of minute red globules. Undoubtedly the fat was absorbed, inasmuch as the animal had been previously starved. And it is evident that it was not absorbed as such but taken up in water-soluble forms

of its constituents. Presumably, the fatty acids entered the villi in the form of soaps: for the villi were stained a diffuse red, which was readily decolorized by weak alcohol, and it is well known that oleic acid stained with this dye is decolorized with much greater difficulty than soaps.

Two Cases of Gonococcic Septicemia Terminating in Cure, and followed by Typhoid Fever.—DIEULAFOY (*Presse méd.*, 1909, xl, 353) reports two cases of gonococcic septicemia following an acute specific urethritis. In the first case the gonococcus was obtained from the urethra and the blood on two different occasions. Gonococcic vaccine was employed as treatment. Recovery occurred after the third injection. The scar of a mitral endocarditis remained. After two days of apyrexia there followed a short attack of typhoid fever with positive blood culture and serum reactions. The second case ran a similar course with blood cultures positive for the gonococcus and B. typhosus in their respective attacks. Dieulafoy remarks upon the infrequency of cure of gonococcic septicemia, and comments upon the possibilities of the pathogenesis of the typhoid infection. Ruling out the possibilities of the gonococcic vaccine being contaminated by typhoid bacilli, of "house" infection in the wards, and the typhoid bacillus being associated with the gonococcus in the first febrile attacks, he considers it most likely that these patients bore the bacillus in a latent state, possibly in the biliary passages; and that the gonococcal septicemia resulted in a condition favorable for their explosion.

Viscosity of the Blood and Iodine.—BOVERI (*Presse méd.*, 1908, lxiii, 546) studied the effect of iodine upon the viscosity of the blood in twelve patients with arteriosclerosis and hypertension. He gave them sajodin, iodipin, and iodogelatin in the ordinary doses, and measured the viscosity of the blood and serum at intervals of ten, twenty, thirty, and forty hours. A very definite diminution in the viscosity of the blood followed in eight of the cases. The serum did not show the same changes as the blood in toto. It remained stationary. The arterial tension fell during the treatment, as much as 65 mm. of mercury (by the Riva-Rocci apparatus) in some instances. The same results were obtained in rabbits by the administration of the iodide of sodium and potassium. The viscosity of the blood was found to be increased or diminished constantly with the increase and decrease in number of red blood corpuscles. In a general way there seems to exist a parallelism between arteriosclerosis, viscosity of the blood, and response to treatment. The greater viscosity is found in the more advanced arteriosclerosis; whereas iodine accomplishes a diminution in viscosity in inverse proportion to the severity of the disease.

The Compensatory Role of Accidental Subcutaneous Emphysema in Two Cases of Pneumothorax with Valvular Opening.—PERRIN (*Arch. gén. de méd.*, 1909, iii, 193) calls attention to the fact that subcutaneous emphysema following the puncture of a pneumothorax is considered a serious complication, especially when the latter is of the valvular type. The emphysema may become generalized and require pleurotomy, or a cellulitis may be set up by extension from the septic pleurisy.

Fortunately, it is not always of such bad prognosis, and the author reports his two cases as evidence of this fact. In both cases the subcutaneous emphysema was produced accidentally by thoracentesis. The puncture wound acted as a veritable safety-valve, compensating for the continual accumulation of air in the pleural cavity by evacuating it into the cellular tissues where it was absorbed. Cure resulted spontaneously. However, Perrin emphasizes the severity of this complication in the majority of cases. The likelihood of the accident is much less if a very fine, sharp needle is used for puncture, so that the opening of the puncture may be easily obliterated. These patients should be watched very carefully in order to interfere if suffocation should be imminent or if the emphysema appears to spread very rapidly. It might be advisable to leave in place a needle with valves as devised by Boinet, which would allow the escape of air but no inflow. Perrin suggests this instrument as useful in the systematic treatment of pneumothorax of the valvular type.

Fixation Abscess in the Grave Accidents of Saturnism.—CARLES (*Bull. gén. de thér.*, 1909, February 8) advances for trial an interesting medication for the more grave accidents due to the accumulation of lead in the organism. By producing a fixation abscess through the subcutaneous injection of turpentine, he hoped to withdraw from the body sufficient lead to make a difference in the symptoms of saturnine intoxication. He carried this out in dogs poisoned with red lead, and from the pus in every instance obtained more lead than in equal amounts of the blood, brain, and liver This was equally successful in a dog poisoned over a period of two months. The clinical application was made in only two cases, one of bronchitis, and one of lead colic. Again, lead was extracted from the pus of the abscess. However, the quantity was small and the clinical results not sure. His explanation is a phagocytosis of the visceral lead by the leukocytes and its transportation to the fixation abscess.

Carnot (*Progrès méd.*, 1909, xii, 159) points out that these results are confirmatory of his own, published in 1894, when he contended that metallic salts in circulation are localized in the region of inflamed organs and that it suffices to produce a traumatism or local suppuration in order to fix the lead salts absorbed. This elective fixation of circulating poisons in the region of inflammatory lesions is comparable to that of microörganisms. It permits the explication of an attack of gout in the region of an injured joint by the elective fixation of uric acid. It explains certain localizations of medicines in the course of local inflammations, as salicylates in rheumatic foci and mercury and iodides in syphilitic lesions. Carnot agrees with Carles that the method may have therapeutic usefulness in a case in which it is urgent to disintoxicate the organism.

Pathological Diagnosis of Mitral Valve Lesions.—SCHABERT (*Deutsch. Arch. f. klin. Med.*, 1909, xcvi, 116) has studied all hearts for the last two years by the "closure test" (Schliessprobe). The wall of the auricle is slit down to the auriculoventricular opening, exposing the mitral valves. An opening is then made in the apex of the left ventricle and a stream of water is directed into the ventricle, while the valves are directly observed.

In this way one can, as a rule, readily differentiate between normal, insufficient, and stenotic valves. Weighing the ventricles separately, according to the method of Müller is also of great aid, and in combined mitral lesions the weights of the ventricles and the closure test allow of closer analysis than has heretofore been possible. The closure test has revealed a surprising number of cases of pure mitral stenosis. Schabert concludes: (1) The closure test is satisfactory in the great majority of cases examined, and permits of far more certain decision as to the presence of stenosis or insufficiency than the usual inspection of the valves. (2) The results of the closure test should be controlled by weighing the ventricles separately. (3) Cases of pure stenosis are only recognizable by the closure test. (4) In pure mitral insufficiency an hypertrophy of the left ventricle is always present.

Myeloid Chloroma.—JACOBAEUS (*Deutsch. Arch. f. klin. Med.*, 1909, xcvi, 7) reports two new cases of myeloid chloroma or chloroleukemia, as Naegeli calls the disease. This brings the total number of myeloid chloromas to ten since Turk's original case in 1903. In both of Jacobaeus' cases myelocytes and myeloblasts were conspicuous in the blood picture. The diagnosis was made antemortem in the first case by the greenish skin metastases, in the second by blood examination; 5 c.c. of blood was treated with citrate and centrifugalized. The resulting layer of leukocytes had a faint greenish tint. Controls from six normal individuals and chronic myeloid leukemias, which were examined similarly, showed no trace of green in the leukocyte layer. Thus, for the first time, examination of the blood alone has led to the correct diagnosis of chloroma, as the postmortem finding subsequently proved.

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY HOSPITAL,

AND

T. TURNER THOMAS, M.D.,

ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL, AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

The Bone Fragility and Eburnation of Rachitis.—BLANCHARD (*Amer. Jour. Orthop. Surg.*, 1909, vi, 616) says that normal bone ossifies slowly. Normal unossified bone bends under the pressure of the osteoclast. Rachitic bone may remain friable indefinitely, but with favoring conditions eburnates suddenly. Eburnated bone breaks hard, and is not amenable to any pressure of iron braces or a plaster of Paris. Very mild bow-leg that is not at all or only slightly rachitic may be autocorrected. Rachitic bow-leg, knock-knee, and anterior bent tibia are never autocorrecting and frequently grow worse in bed. Badly deform-

ing, rachitic knock-knee and bow-leg are occasionally seen in children that have never walked. Rachitic bow-leg predisposes to rachitic coxa vara. Rachitic knock-knee predisposes to rachitic coxa valga. Knock-knee is distributed distortion of all the long bones of the legs, having its usual inception in an inbend of the upper tibial shaft. Bow-leg is a distributed distortion of all the long bones of the leg, having its usual inception in an outbend of the femoral shaft, though, as the deformity progresses, the secondary outbend of the tibia frequently becomes the most pronounced. The skiagraphic observations of a large number of cases would seem to prove quite conclusively that these deformities closely follow fixed lines of development. Rapid osteoclasis is the safest and quickest means of correcting most of the rachitic deformities of the legs. The term rapid osteoclasis is used because osteoclasis that is not rapid is always a failure and brings the osteoclast into disrepute. These conclusions are drawn from nearly 1000 cases of knock-knees, bow-legs, and anterior bent tibiæ corrected by rapid osteoclasis with the Grattan osteoclast.

A New Method of Treating Hydrocele.—Marcozzi (*Ann. d. mal. org. gén.-urin.*, 1909, i, 739) says that the tunica vaginalis is important for the function of the testicle. A certain number of hydroceles, especially those of recent origin, show only slight distention and thickening of the tunic, and for this reason have a tendency to cure. In such cases it would be best to try first some non-operative treatment, such as the injection into the sac of some suitable medicament, like iodine or silver nitrate. It does not always cure, but is frequently followed by recurrence. This is probably due to the fact that the effect of the injected substance is too fleeting to permit it to act long enough upon the chronically inflamed serous sac. Marcozzi has employed in 10 cases an injection of fragmented magnesium thread. This is first washed in ether and then sterilized in distilled water. After being injected it is easily absorbed and produces a durable stimulation and reaction of the serous surfaces. As the result of his success in the use of this method, he concludes that it is more advantageous than all others. It produces no pain and permits the patient to continue at his usual occupation. There is no risk of necrosis of the scrotal tissues, such as has followed the employment of the tincture of iodine.

Partial Sequestration of a Transplanted Piece of Bone.—Axhausen (*Arch. f. klin. Chir.*, 1909, lxxxix, 281) resected the upper end of the humerus and the joint capsule for a secondary cancer in this situation, which had followed an operation for the removal of a cancer of the breast, two and one-half years before. There was no recurrence in the scar or in the lymph glands. After removal of the upper end of the humerus, the second, right metatarsal bone was extirpated and its attached muscles carefully removed. Then several incisions were made through its periosteum and its proximal end was freshened and pointed. Its pointed end was then driven into the medullary cavity of the upper end of the divided humerus. The two attached bones were then replaced so that the cartilaginous surface of the metatarsal bone fitted into the glenoid cavity of the scapula. The muscles, which had been detached from the humerus, were sutured to the periosteum of the upper divided

end of this bone and the wound closed with the introduction of a small drain. Moderate infection followed, but although the transplanted bone was bathed in pus, it did not become entirely necrotic, but was only partially sequestered. Recurrence of the carcinoma in the humerus made another resection necessary, so that a histological examination was possible. This showed that the original transplanted bone substance had died completely, and that the greater part of the periosteum had lived and had given rise to abundant bone formation. Some of the medullary substance also remained, especially in the region of union, where there was considerable callus formation. The new bone which had taken the place of the dead bone almost completely, came only from the periosteum and medullary substance. Lacunar resorption and apposition played an important part in the process. In the presence of a moderate infection, although the whole transplanted bone becomes necrotic, the greater part may be retained and only a part be extruded as a sequestrum, that is, only a partial sequestration of the total necrotic bone occurs.

A Case of Stasis Cyanosis following an Epileptic Seizure, Simulating Traumatic Asphyxia.—ALEXANDER (*Annals of Surgery*, 1909, xlix, 762) reports the case of a young man who fell to the floor in an epileptic attack and was taken to the hospital. At the time of the fit it was observed that his face was blue, that his eyes were bloodshot, and that he remained unconscious several minutes. On examination at the hospital, his face and neck were found to show a diffuse, bilateral, bluish, and slightly punctiform discoloration of the skin, that disappeared but slightly, if at all, on pressure. This discoloration stopped with a decided line of demarcation just where the tight collar passed around the neck. Below this line the skin was of a normal color. The eyes showed a marked subconjunctival ecchymosis, more marked on the left. The chest and abdomen failed to show any signs of recent injury. Fourteen days after the accident the discoloration had almost disappeared. Alexander thinks that the factors producing this condition are similar to those producing traumatic asphyxia, namely, a fixed thorax, a closed epiglottis, an increased intrathoracic pressure, a lack of aëration of the blood, and the incompetent and absent valves of the jugular, subclavian, and facial veins.

Pancreatic Surgery.—ROSENBACH (*Arch. f. klin. Chir.*, 1909, lxxxix, 303) says that in recent years we have gained a more exact clincial knowledge of pancreatitis and pancreatic necrosis, and, particularly, we have learned to estimate more correctly their relation to diesases of neighboring organs, as the gall-bladder and stomach. We have also advanced considerably in our knowledge and successful treatment of wounds of the pancreas. Isolated subcutaneous injuries are rare. Up to 1905 only 8 uncomplicated cases had been reported, and all these, whether operated on or not, died. Garré, in 1905, while operating for what he thought was a ruptured intestine, found a transverse tear of the pancreas, which he sutured and which was the first cure. Since then Heinecke collected 10 cases, 8 of which were cured by operation, and more recently 8 other cases have been reported, of which 4 were saved by operation and 4 died. Rosenbach reports an additional case, which recovered after operation. A woman, aged forty-seven years, was struck in the abdomen by the pole

of a wagon, and fell unconscious. On recovering consciousness, she complained of severe pain in the abdomen especially in the region of the stomach. Soon there was vomiting of bile, without blood, and for three days there was passage of flatus, but no feces. Distention of the abdomen was marked, especially in the epigastrium. The recti were rigid and the whole abdomen very tender. Above the umbilicus was detected a feeling of resistance not well defined. The whole clinical picture suggested an intra-abdominal injury, probably of the stomach. On opening the abdomen an encapsulated mass, about the size of an apple, was found below the liver and above the stomach. After separating some adhesions a brownish red, thin fluid escaped. On raising the omentum and transverse colon there was seen near the spine, in the mesocolon, a mass of yellowish gray necrotic material. Many areas of fat necrosis were seen in the omentum and mesentery. An incision was made through the gastrohepatic omentum, that is, above the stomach, into the above-mentioned encapsulated mass. A quantity of brownish red old blood, but no fresh blood escaped. No tear in the pancreas was detected by the finger, and an exact inspection was impossible. The wound was tamponed and drained. Frequent examination of the urine showed no sugar. From the wound there was an abundant discharge, which upon examination was recognized as pancreatic secretion. It required nearly four months for the fistula to close.

Traumatic Asphyxia.—DESPARD (*Annals of Surgery*, 1909, xlix, 751) says that the term traumatic asphyxia has been applied to the series of phenomena following the suspension of the respiratory function for a more or less prolonged period by forcible compression of the thorax or abdomen, or both. These consist of the usual symptoms of suffocation, associated with a peculiar bluish mottling of the skin of the face and neck, sometimes extending to the upper part of the thorax, and occasionally to one or both arms, accompanied by subconjunctival hemorrhages, and frequently bleeding from ears, nose, and pharynx. He reports the case of a man who was compressed by a moving elevator for about five minutes. The line of compression extended from the left side of the base of the neck to the right lumbar region. When found his face was said to have been almost black, eyes bulging, mouth open, and tongue protruding, and he was completely unconscious. Examination later showed both arms to be apparently paralyzed. Over the forehead, face, and neck, and extending to the right shoulder anteriorly and posteriorly, were what seemed to be small ecchymotic spots, dark red in color, which did not disappear on pressure and were separated by normal skin. Gradually the patient recovered, so that in about three weeks after the accident he had so far recovered as to be permitted to leave the hospital, to return to the out-patient department for electrical treatment. Six weeks later he had fairly recovered the use of the right arm, but there was still some muscular weakness. In this form of suffocation not only is the air prevented from entering the lungs by their inability to expand, but the contents of the thoracic vessels may be forced out and, in the case of the veins, the current is reversed. The valves are then overcome and the blood is dammed back into the capillaries. If the force acting is sufficiently great, it is conceivable that the capillary vessels would be dilated to a point where paresis would ensue.

The Technique of Implanting the Ureter into the Intestine.—Ssimzemsky (*Zentralbl. f. Chir.*, 1909, xxxvi, 706) says that experimental attempts to implant the ureter into the large intestine have given unfavorable results. Mirotworzew reported that the implantation on both sides in dogs was fatal in all his cases, and when only one ureter was implanted more than half of the dogs died. Of 28 patients operated on by Tichow, 9 died; and both patients operated on by Diakanow also died. Yet the indications for operation are so pressing that undoubtedly these cases will continue to be operated on in the future. Therefore, perfection of the technique should be sought. The usual causes of death are ascending pyelonephritis and urinary peritonitis. Ssimzemsky offers the following technique: The ureter is divided at a convenient situation on the posterior wall of the small pelvis, and without isolating it from the surrounding tissue, it is taken up with small flaps of the overlying peritoneum and the attached connective tissue to the extent of about 6 or 7 cm. Then a suitable forceps is introduced into the rectum and the wall of the intestine at the site of the anastomosis is made to protrude on the points of the forceps. A small incision is made over this prominence, the forceps is protruded, the divided end of the ureter seized and drawn into the bowel about 1 to 1.5 cm. The flaps of peritoneum covering the ureter are then applied to the serous surface of the intestine around the seat of anastomosis. Two stitches fix the ureter in the small intestinal wound and grasp the serous and muscular coat of the ureter and surrounding cellular tissue and the serous coat of the ureter. The peritoneal flaps of the ureter are then sutured to the serous surface of the intestine. After such an anastomosis on the cadaver, the kidney was isolated, the pelvis of the ureter opened, and water under considerable pressure forced through the ureter into the intestine. After the water had somewhat distended the intestine it escaped through a tube introduced into the rectum. None escaped into the abdominal cavity.

The Operative Treatment of Old Empyema.—Goldmann (*Zentralbl. f. Chir.*, 1909, xxxvi, 785) calls attention to the fact that the Schede operation requires a free exposure of the whole cavity of the empyema, and above all the removal of all bony covering. The Delorme decortication of the lung fails in many cases, and in some the removal of the thickened pleura must be combined with the classic Schede resection, in order to obliterate the cavity. He concludes that the ideal healing will be promoted the more rapidly: (1) The more completely the overlying chest wall is resected and the thickened pleura is extirpated; (2) the more thoroughly are our efforts made to cause the decorticated lung to expand; and (3) the more quickly we bring about the permanent unfolding of the lung by causing a prmiary adhesion of the lung to the overlying soft parts. He reports the case of a young woman, who had a pneumonic empyema June 2, 1906. On June 28 a rib resection (2 ribs) was done. The wound was healed November 9, but later broke open again. April 11, 1907, another resection was done (7 cm. of one rib) on account of the persisting fistula. Later a Schede operation was performed and forced expiratory gymnastics carried out, without success. After determining, by probing, the presence of a cavity about the size of an apple, Goldmann, on May 6, 1908, freely exposed this cavity, removing the sixth rib. He then removed the lining of the chest wall and the lung pleura.

The wound was contracted by sutures until there was left only a small drainage track. August 10 the fistula was reopened and the lining wall of the now smaller cavity was again removed. This was repeated November 30, and the lung was now drawn out by a forceps and fixed by sutures to the margins of the cavity. A flap was made from the overlying skin and soft tissues with its base toward the spine and drawn over the presenting lung tissue. For a few days there was high fever. Six days after the operation there was evacuated from the upper angle of the wound a large quantity of almost clear exudate, free of bacteria. Then the temperautre fell and the wound closed firmly. Over the whole area of the old cavity, vesicular breathing could be heard. There was a good curve to the thorax, with no sinking in of the skin and no scoliosis.

The Utilization of the Coagulation Time of the Blood in Surgery.—DENK and HELLMANN (*Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1909, xx, 218) say that the Wright method of determining the coagulation time of the blood, with careful attention to sources of error, is the most reliable method of determining comparatively slight deviations from the normal, which may be regarded as pathological. The practical application of this examination is of great value to surgeons in detecting hemophilia, even of the mildest grades. Operations on bleeders should be undertaken only after prophylactic treatment with calcium salts or serum injections, when the effects of these substances are to be controlled by the determination ot the coagulation time of blood. Operations that are not urgent should be declined in severe cases of hemophilia, in which the clotting time is higher than normal in spite of preliminary treatment. In the after-treatment of operations, in which the coagulation time is normal or accelerated, milk and all other foods which hasten coagulation should be avoided. In such cases citric acid should be given in large doses, on account of the possible relationship between rapid coagulation and emboli formation. Milk should be replaced by foods lacking in calcium and, therefore, having no effect on the time of clotting.

Thyroid Gland and Vascular Surgery.—CARRELL (*Surg., Gynec., and Obst.*, 1909, viii, 606) says that among the operations able to modify the functions of the thyroid, the ligature of the arteries alone has been used in clinical surgery for the treatment of exophthalmic goitre. Other diseases of the thyroid, especially the "formes frustes" of hyperthyroidism and hypothyroidism may be improved by surgical modifications of the circulation of the gland. It is very logical to suppose that we can stimulate or diminish the activity of a gland by increasing or diminishing its circulation, and perhaps also modify its pathological reactions. But it is premature to think at present of clinical applications along these lines. The study of the question is still at its beginning and a great many of more precise experiments have still to be done. The treatment of hypothyroidism and athyroidism by the transplantation of the thyroid is more immediately promising of practical results. The feasibility of the operation has been demonstrated. The actual problem is to render it ordinarily successful. Success in transplantation depends on two factors, biological, and surgical. We are still ignorant of the biological factors of success and failure in transplantation of tissue between animals

of the same species. We do know exactly what are the interactions of the organism and its new organ. It is certain that, at least, in some cases, the gland adapts itself to its new owner, does not undergo any marked histological changes, and functionates normally. But we do not know how frequent are the cases in which the new tissue and its host will have a pernicious influence on each other. The surgical factors can be probably controlled. It must be hoped that the technique of the graft by simple implantation will be improved. The operation is very much simpler than the transplantation with anastomoses of the vessels. However, if it is found that it cannot frequently be successful the transplantation of large pieces of thyroid with union of their vessels to the vessels of the patient must be attempted. In this indirect manner vascular surgery may improve the results of the treatment of hypothyroidism and athyroidism.

THERAPEUTICS.

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF APPLIED THERAPEUTICS IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

The Physical Treatment of Bronchial Asthma.—STRASSER (*Monats. f. d. physikal.-diät. Heilmethod*, 1909, i) has made use of hydrotherapeutic measures for the treatment of asthma for sixteen years. These measures include general douches, local douches applied to the legs, simple and Scotch douches to the epigastrium, etc. Strasser has had good results from their use even in severe cases complicated by bronchitis. They all diminish the reflex nervous irritability, which seems to play such an important role in the etiology of asthma. Von Strümpell has advocated the use of electric light baths for the treatment of asthma, and Strasser confirms his view regarding their beneficial action. Von Strümpell has his patients remain in bed for two hours after the electric light applications. Strasser prefers the use of some of the milder hydrotherapeutic measures following the electric light baths. He does not think it advisable to allow the patients to remain for two hours bathed in perspiration. In mild cases, Strasser follows electric light bath of ten minutes' duration with a douche or light rubbing down and very light exercise. In severer cases the electric light baths are made longer in duration and the hydrotherapeutic measures following are milder. The patients are then made to rest in bed for one or two hours. Strasser regulates the length of the baths by the subjective condition of the patient. As a rule, the initial bath is not more than six or eight minutes in duration, and, subsequently, never more than fifteen minutes. Daily baths are sometimes exhausting, and if so, they should be given every other day, or on two days out of each three. On the intervening days the treatment is intermitted or general mildly tonic hydrotherapy is given. Von Strüm-

pell does not recommend the baths at the height of the attacks, but Strasser has found them very useful even in acute attacks. Strasser agrees with v. Strümpell that probably the most important action of the baths is the diaphoresis induced by them. He thinks it also may be true that the electric lights have a specific action upon the bronchial mucous membrane, increasing the secretion from the glands and epithelium. By virtue of these actions they may aid in the elimination of the toxic substances which are the inciting agents of asthma. Strasser also suggests that they may have a direct antispasmodic action upon the skin and internal organs. The effect of heat upon various hysterical spasmodic affections is well known, and Strasser bases his theory upon this premise.

General Principles in the Treatment of Diseases of the Heart.—Barr (*Brit. Med. Jour.*, 1909, i, 989) introduces his paper by discussing the preventative treatment of cardiac disease occurring as a result of the acute infections, particularly rheumatism, or as a result of the degenerative lesions of the heart and bloodvessels so common in later life. He deprecates the tendency for the indiscriminate use of cardiac tonics in heart disease. He says that many of the cardiac tonics cause an increase of arterial pressure which, if long maintained, leads to irreparable damage to the aorta. He believes that measures which lessen the work of the heart are of much greater value than cardiac tonics. When the balance between the two sides of the heart is well maintained drugs are usually unnecessary. Among the measures which lighten the work of the heart he mentions rest, graduated exercise, reduction of intake of fluids, reduction of amount of food, sweating, diuresis, use of cathartics, and various hydrotherapeutic measures. With regard to exercise, Barr does not prefer any routine system, but believes that the exercise must be fitted to the individual. Exercise increases oxidation and hastens the elimination of waste products that are the main causes of arterial degeneration. Properly graduated exercise increases the tone of the cardiac muscle and at the same time increases the general muscular capacity. If the blood pressure is high, Barr prefers the use of decalcifying agents and mild cathartics over long periods of time in order to maintain the pressure at a constant lower level. If there is any tendency to thrombosis or embolism, he believes that decalcifying agents, such as phosphoric and citric acids and their soluble salts, should be used. In cases in which there are urgent symptoms indicating cardiac thrombosis or pulmonary embolism, strong ammonia should be freely used. Barr gives a mixture of five minims each of liquor ammoniæ fortior and spiritus chloroformi in a wineglassful of barley water every half-hour or hour for this purpose. He states that a free hemoptysis in a case of mitral stenosis is often the best form of bloodletting to relieve the distended right heart, and, therefore, it should not be checked too quickly. He condemns the use of hemostatics and advocates the use of nitroglycerin and atropine. In all of the spasmodic affections associated with a high arterial tension, such as angina pectoris, the best remedies are nitroglycerin, morphine, atropine, amyl nitrite, and the iodide preparations. Barr says that, so far as we know, the presence of free calcuim ions in the blood and tissues is necessary for effective muscular contractions. He cites Bell's work, who showed that when a solution of calcium chloride was injected into the veins of a rabbit, the rhythmical

contractions continued and their amplitude increased while their frequency diminished. Barr believes that a renewed supply of calcium ions is indicated in febrile conditions because of the increase of their excretion in such conditions. An inordinate supply of calcium ions increases the viscosity of the blood, raises the blood pressure, hastens the formation of scar tissue, and leads to degenerative changes in the heart and bloodvessels. When the supply of calcium ions in the blood is too great, Barr states that the cardiac contractions tend to become slow though still effective. Because of the greater muscularity of the left ventricle, there then occurs an asynchronous action of the right side of the heart with consequent irregularities and intermissions. Therefore, in such conditions as Stokes-Adams disease Barr advises an elimination of the lime salts and the use of decalcifying agents.

Salt-poor Diet as a Therapeutic Measure.—MENDEL (*Münch. med. Woch.*, 1909, ix, 433, and x, 516) believes that the good effects of many of the dietetic methods for the treatment of circulatory disturbances and obesity are to be ascribed simply to the low sodium chloride content of the diets used. Thus, he explains the beneficial effects of the Karell milk cure with its various modifications. Mendel has obtained the same results by the use of a mixed diet low in sodium chloride content. He reviews some of the recent experimental work and clinical observations of the results obtained by the use of salt-poor diet in various affections. He mentions particularly Finklestein's results in the treatment of eczema of children by such a diet. Mendel says that even normal amounts of salt in the diet of healthy individuals cause a retention of blood in the body of from 1.5 to 3 liters. An excessive intake of salt may cause a marked retention of water even in the healthy individual with sound kidneys. However, in normal individuals, there is no trausudation or exudation of fluid. Mendel cites various animal experiments performed by Cohnheim and Lichtheim, who found that normal animals could eliminate large quantities of salt solution introduced intravenously without the production of any œdema. If, however, they produced an artificial inflammation, a marked œdema always occurred in the neighborhood of the inflammation. Consequently, there must be some abnormal condition of blood or lymph vessels before œdema can be produced. They were also able to produce œdema in case they prevented the venous return by a ligature. Mendel believes that retention of salt with consequent retention of water not only alters the clinical picture of inflammation, but has an injurious action on its course. This effect, he believes, is more striking in various skin affections and in inflammations of serous membranes. He believes that an excess of salt increases the accumulation of fluid in pleurisy and in synovitis. These accumulations of fluid compress the lymphatics and capillaries, and thus prolong the course of the inflammation. Mendel describes some very favorable results obtained by the reduction of salt in the diet of various affections. The cases he treated in this manner comprise cases of burn, of eczema, of ascites with cirrhosis of liver, of pleurisy, of gonorrhœal epididymitis, of joint affections, of venous thrombosis, and of arteriosclerosis. He states that in parenchymatous nephritis, the most notable results can be obtained by a salt-poor diet. A salt-poor diet relieves peripheral resistance in heart affections, increasing the amount of urine, and thus reliev-

ing stasis. Mendel says that it is not necessary to exclude all salt, but to reduce the amount to from 30 to 60 grains a day. He has found that a milk diet is the simplest and easiest form of a salt-poor diet.

The Results of Drug Treatment in Five Hundred Cases of Delirium Tremens.—RANSON (*Jour. Amer. Med. Assoc.*, 1909, lvi, 1224) summarizes his conclusions with the use of drugs in the treatment of 500 cases of delirium tremens as follows: (1) In incipient cases the patients respond readily to treatment with chloral, ergot, bromides, and whiskey, the drugs being mentioned in the order of their value. (2) Delirious patients are very resistant to treatment. In the cases studied the administration of the sedative drugs increased the mortality. This was most evident when scopolamin was used, that drug increasing the mortality 13 per cent. These unsatisfactory results with the sedative drugs were due to the large doses used. It will be shown that small quantities, for example, 15 to 30 grains of chloral in twenty-four hours, may be given with good results, but that when larger doses are given the death rate increases with the amount of sedative administered. (3) The only drug which reduced the mortality was ergot. By its use the death rate was reduced 21.6 per cent. (4) When whiskey was given the mortality was increased 1.8 per cent. The 500 cases which serve as a basis for Ranson's article were treated at the Cook County Hospital, Chicago, over a period of three years. They were divided into two classes, incipient and delirious. The incipient cases were those which showed only insomnia, restlessness, and tremor. The fully developed cases were noisy, delirious, and had well-defined hallucinations. Of the sedative drugs the bromides were most extensively used. A mixture of sodium, potassium, and ammonium bromide, in doses of from 10 to 30 grains each every four hours, was used in 198 cases with delirium. This series showed a mortality of 45.5 per cent. In 103 similar cases treated without bromides, the mortality was 40.8 per cent. In 190 incipient cases treated with bromides, only 32.6 per cent. of the patients became delirious; while of 156 similar cases not treated with bromides, 52.8 per cent. of the patients became delirious. Chloral was also extensively used and frequently was given in combination with the bromides. Ranson believes that it is of no value after active delirium has set in. In fact, it seemed to increase the mortality by 2 per cent. However, he believes that chloral is of great value in the treatment of incipient cases. It reduced the percentage of cases developing delirium in the hospital from 48.9 per cent. to 20.6 per cent. Ranson believes chloral superior to the bromides in the treatment of incipient cases because of the greater speed of its action. He found morphine of very little value, having no effect at all in delirous cases and far inferior to small repeated doses of the other sedatives in the incipient cases. Scopolamin was given in single doses of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain to a series of incipient cases. Like the morphine series, this series of cases seemed to show that single doses of sedatives have no controlling action upon the incipient cases. In addition, Ranson found that scopolamin was extremely dangerous when given to actively delirious cases. He speaks particularly of the good effects of ergot in the treatment of these cases. Ergot was given in the form of the fluid extract, in dram doses, repeated every four hours, and often in combination with whiskey. In those cases in which ergot was used the mortality was only 30 per cent.,

while among those patients treated without ergot it was 51.6 per cent. Ranson says that this result is striking when we consider that usually the sedatives increase the mortality. Ranson likewise found ergot of value in the incipient cases, the percentage of patients developing delirium being reduced 23.3 per cent. by its use. He says that any explanation of its action would be pure speculation, but speaks of the possibility that it may decrease the cerebral hyperemia. Ranson also believes that whiskey is of use in the treatment of incipient cases, lowering the percentage of patients becoming delirious by 20.2 per cent. In the delirious patients, he believes it is useless.

The Pathogenesis and Therapeutics of Diarrhœa.—A. SCHMIDT (*Med. Klin.*, 1909, xiii, 457) reviews briefly some of the causes of diarrhœa, and mentions particularly a form with serous, foul-smelling stools. This form, he believes, is due to an abnormal secretion of an albuminous fluid which putrifies readily. He discusses briefly the treatment of the different forms of diarrhœa, classifying the remedies into the opiates, the astringents, and the intestinal antiseptics. The opiates are believed to be beneficial by reason of their antiperistaltic action, and consequently they are used in cases of diarrhœa due to increased peristaltic action. Schmidt believes they should be used only in cases of chronic diarrhœa when all other measures fail, never for acute cases. He believes that the opiates are an example of symptomatic therapy and have no relation to rational therapeutics. The use of the astringents rests upon a much firmer etiological basis, but their application is limited to certain forms of diarrhœa, *e. g.*, those showing signs of a subacute or chronic inflammation of the mucous membrane. Schmidt says that he has made many clinical observations and experiments in order to determine the most efficient intestinal antiseptic, and he believes that hydrogen dioxide is the most efficient and least harmful. He found that pure agar-agar was able to take up from 10 to 12 per cent. of hydrogen dioxide, and that it yielded it up very slowly in the form of nascent oxygen. He used this hydrogen-dioxide-agar for the treatment of diarrhœa with serous foul-smelling stools, with good results. It had no apparent effect upon diarrhœas originating in the large intestine or those due to intestinal tuberculosis. Schmidt concludes by saying that since the hydrogen-dioxide-agar is perfectly harmless, it should be more widely used, in order to determine its further uses and limitations.

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D., AND THOMPSON S. WESTCOTT, M.D.,

OF PHILADELPHIA.

Staphylococcic Infection Treated with Killed Bacteria.—SOPER (*Pediatrics*, xxi, No. 3) reports observations on the application of opsonic therapy to bacterial infections in infancy and childhood. The

majority of his cases were furunculosis in children, ranging in age from two months to seven years. In each case the staphylococcus was isolated before treatment was begun, and no medicinal treatment was used. The opsonic index was not followed. Twelve cases received on an average two inoculations each; 6 received three, and 2 cases one each. Most of the cases were cured in from one to four weeks; a few were not improved. A bacterial emulsion was prepared in the following way: a twenty-four-hour growth of pure culture on plain slant agar was washed with 3 c.c. of sterile normal salt solution and placed in a water bath at 65° C. for one hour. The number of cocci per c.c. was estimated by the Wright method of standardization, or by counting in a Thoma-Zeiss red blood counter, using a solution of methylene blue as a stain. Sterility of the emulsion was proved by inoculating with it a tube of slant agar and incubating for twenty-four hours at 37° C. If no growth appeared, it was considered sterile enough for subcutaneous inoculation. Using the same amount of salt solution and the same sized stroke on the agar produced fairly constant quantities of bacteria per c.c; 0.5 to 1 c.c. usually gives a dosage of 50,000,000 to 100,000,000. Local inflammatory reaction was slight or absent. Comparison of stock vaccines with autogenous showed little difference in behavior, autogenous vaccine curing each of 5 cases, and stock vaccine 75 per cent. of 15 cases. Usually a small dose, 30,000,000, was given at first, and later the dose was increased up to 100,000,000. In some of the older children 250,000,000 were used at first and increased to 500,000,000.

Teratoma of the Testicle.—LORRAIN (*Bull. et mém. de la soc. anat. de Paris*, 1909, lxxxiv, 65) reports the case of a boy, aged fifteen years, whose left testicle had been noticed to be enlarged since he was six months old; it grew slowly until his ninth year, at which time he had measles. Since then it had grown more rapidly. At the time of the report the right testicle was of normal size, the left twice as large. At the base of the testicle was to be felt a hard mass, regular in outline, not transparent and slightly tender; the epididymis was intact. The diagnosis rested between tuberculosis and cyst. After removal of the testicle it was noticed that the testicle and the tumor were separated from each other by a constriction, the tumor mass, however, extending into the interior of the testicle. The mass contained sebaceous material, glands, and four teeth, thus showing the tumor to be a teratoma.

The Distribution of Tuberculous Lesions in Infants.—MARTHA WOLLSTEIN (*Archiv. Int. Med.*, April, 1909) has investigated the manner of infection and the localization of tuberculous lesions in children, by analyzing the autopsies of 185 children showing tuberculosis. These children came from unhygienic tenement districts in New York and were all under three years of age. From this series of cases it appears that of all infants under one year coming to autopsy, 12 per cent. showed tuberculous lesions; 36 per cent. of those between one and two years, and 32 per cent. of those over two years. Of infants under three months only 1.8 per cent. had tuberculosis. In these 185 cases there were 13 in which the lesions were limited to the respiratory tract, and only 1 case involving the intestines alone. The point of entrance of the bacillus

was clearly 13 times in the lungs and once in the intestinal tract. Four additional cases were of undoubted intestinal origin, 40 of respiratory origin, and one of mixed origin. In 8 cases the mesenteric lymph nodes were involved without a lesion in the intestines, but with marked pulmonary involvement. The ulcers in the intestines were most frequent and advanced in the lower ileum. In the kidneys young miliary tubercles were found in 67 cases, usually in both kindeys. The pulmonary lesions varied from discrete miliary tubercles to cheesy areas and cavity formation. The right upper lobe was involved oftener than the other lobes. This study of cases under three years shows the rarity of undoubted cases of intestinal tuberculosis, and that tuberculosis of respiratory origin predominated over that due to ingestion of the bacillus. The tendency of the tuberculous process in lymph nodes in young children is toward progressive degeneration rather than healing, as caseation and suppuration were found to be the rule in these cases.

Gonorrhœa in Little Girls.—SCHEUER (*Wien. klin. Woch.*, 1909, No. 18) reports 39 cases of gonorrhœa in little girls. In 15 cases the urethra was involved. In no case was he able subsequently to determine any involvement of the pelvic peritoneum or adnexa. Treatment consisted in rest in bed and sitz baths. The vagina was washed out three times a day, with 0.5 to 2 per cent. protargol solution or with a 0.1 per cent. ichthyol preparation. In treating the urethritis he employed thin protargol bougies. The course of the disease was usually found to be chronic, and in many cases a cure was not obtained until after three months of treatment.

The Cutaneous Test for Tuberculosis.—HERRMAN (*Pediatrics*, 1909, xxi, No. 3) discusses the preference for the cutaneous test. He states that while a positive reaction does not necessarily show the presence of active tuberculous lesions, this test is simple, universally applicable, and free from danger. Autopsy findings prove it as trustworthy as any of the tests recently employed. It is more valuable in infants and children than in adults, and a positive reaction more significant, because there is less likelihood of a healed tuberculous lesion being present. He notes the objections to the various tests. Injections of tuberculin are painful; can be used only when there is no fever; and the temperature must be taken at short intervals. The puncture test is painful and has no advantage over the cutaneous test. The conjunctival test is not without danger, and requires a normal eye and conjunctiva. It requires a specially purified preparation of tuberculin, and in infants the tuberculin may be conveyed to the opposite eye by rubbing of the eyes. Doubtful reactions are frequent. Inunctions of tuberculin require a special preparation, and results are not in any way more reliable. Advantages of the cutaneous method are simplicity, ease of application, and freedom from danger; it requires no special preparation, crude tuberculin being used. Patients need not be reëxamined for twenty-four or forty-eight hours. Pain caused by scarification is slight. He uses the following method. The forearm is cleansed and two new sewing needles are held together and dipped into tuberculin (undiluted); a small quantity is thus held between the needles by capillary attraction. Two short scarifications are made at right angles and the tuberculin adhering to the needles is rubbed in, or the drop is placed on the skin first, and scarification made through

it. New needles are used for each patient, and a separate applicator is unnecessary. No control scarification is used. If the reaction is doubtful it is of no value. Herrman has never seen a control which could be mistaken for a positive reaction.

A Case of External Masculine Pseudohermaphrodism; Sarcoma of the Ovary.—BROCA (*Bull. et mém. de la soc. de chir. de Paris*, 1909, xxxv, 294) reports the case of a boy, aged seven years, looking like a boy of ten. There was very little of feminine facies and characteristics about him. He had an abdominal tumor, which had existed for six months. During his sixth year the following surprising condition developed: the penis became quite large, the voice deepened; he developed pubic hair; and his breasts became as large as those of a girl thirteen to fourteen. He had a hypospadia; the left testicle was missing from the scrotum; on the right side a hernia was noted containing a mass resembling a testicle. The abdominal tumor resembled a uterine or ovarian fibroid, but as there was no doubt as to the sex of the patient, it was believed to be a fibroid of the mesentery. The operation revealed a solid ovarian tumor with a normal tube and an atrophic uterus. A fibrous cord represented the vagina. The uterus and left ovary were removed; the right ovary and tube were contained within the hernia, and had no uterine connection. A radical operation was performed for the hernia, the ovary and tube having been reduced into the abdominal cavity. Pathologically the tumor was found to be an endotheliofibromyxosarcoma of the ovary. The penis, as stated above, was well developed; it had a normal glans and prepuce, the urethral orifice being situated 3 cm. back of the glans. A cord passing along its under surface caused an incurving of the glans. The pubic hair was long and very dark. The scrotum was of normal appearance, excepting that instead of hanging below the penis, it surrounded it, the latter seemingly growing out of it, as is the case with the clitoris and the labia majora. There was no sign of vaginal orifice. Four years later the child was still in excellent health; there was no sign of menstrual congestion; the child was of normal stature. The pelvis and thorax presented the characteristics of a male; the breasts were much less developed in proportion than at the time of the operation—in fact, they look like those of a boy. The external genitalia were as before, and there was no sign of a recurrence of the tumor. He was at the head of his class, and was able to take part in the games of boys of his age.

Adenoids, Nocturnal Incontinence, and the Thyroid Gland.—WILLIAMS (*Lancet*, 1909, i, 1245) had the adenoids of a nine-year-old boy removed, hoping it would cure him of nocturnal incontinence. It made the boy much worse, however, and believing that the removal of the adenoids deprived the boy of a necessary internal secretion, he then gave him thyroid extract, one-half grain twice daily. The result was instantaneous and complete, the boy no further wetting the bed. Twenty-four other cases were thus treated, with but one failure. He concludes that adenoid vegetations should no longer be considered as a cause of nocturnal incontinence; in fact, Williams believes that adenoids afford some measure of protection against this weakness, and he further believes that if the two co-exist, it is due to a common cause—insufficiency of the internal secretion of the thyroid gland.

OBSTETRICS.

UNDER THE CHARGE OF
EDWARD P. DAVIS, A.M., M.D.,
PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Pain and Hemorrhage as Symptoms of Ectopic Gestation.—EDEN (*Brit. Med. Jour.*, April 17, 1909) reports eight cases of ectopic gestation, and calls attention to the importance of pain and hemorrhage as symptoms. He calls attention to the fact that symptoms associated with ectopic gestation arise not directly from the presence of the growing ovum, but from secondary lesions, traumatic or inflammatory. These are bleeding from tubal abortion, or rupture, the blood passing into the peritoneal cavity, or bleeding into the Fallopian tube, causing acute distention, the abdominal end remaining closed, or slowly progressive, or recurrent hemorrhage leading to encysted collections of blood, or infection of the gravid tube, or some encysted collection of blood leading to suppuration. He calls attention to the importance in diagnosis of hemorrhage from the uterus, the discharge being dark and persistent in character, as a symptom of ectopic gestation. When free bleeding occurs into the peritoneal cavity, the symptoms are acute abdominal pain, fainting, and constitutional signs of hemorrhage, and shock attended by vomiting, gradually improving, usually occurs after such hemorrhage, which differentiates these cases from perforation of the stomach or intestine, in which the poisonous contents, escaping through the point of rupture, cause the patient to grow steadily worse. The intense abdominal pain seen in ruptured ectopic gestation is caused not by the bursting of the Fallopian tube, but by irritation of the peritoneum by extravasated blood. Recurring attacks of intense pain indicate repeated intraperitoneal hemorrhage. Attacks of acute abdominal pain may also follow tubal abortion, and Eden reports a typical case, in which, before operation, it was thought that the pain arose from the bursting of a pyosalpinx and the escape of its pus into the peritoneal cavity. Pain and nervous disturbance resembling that following intraperitoneal hemorrhage may occur when a Fallopian tube is pregnant without rupture. In a case reported by Eden, a firm, elastic swelling was found in Douglas' cul-de-sac, the patient suffering from shock and abdominal pain. Her pulse, however, was good; her temperature very little disturbed, and the cardinal signs of dangerous hemorrhage were lacking. At operation an unruptured gravid tube was found and readily removed. An encysted collection of blood or hematocele is often found in those cases in which the patient has irregular uterine hemorrhage, with pain in the abdomen or pelvis, this phenomenon recurring at irregular intervals over a considerable period of time; in the writer's case a peritubal hematocele surrounding the left tube was present. In these cases it is not sufficient to put the patient at absolute rest to prevent progressive hemorrhage and the enlargement of the hematocele; operation only will permanently check slow and insidious bleeding. In some cases of pregnancy in a bicornate uterus,

where the ovum lodges in one side of the womb, it develops to a considerable extent and may rupture without hemorrhage. The writer describes a five months' pregnancy in the left cornu of the uterus, where rupture occurred and partial extrusion of the fetus and retention of the placenta. Very little free blood was found in the peritoneal cavity. Eden calls attention to the fact that the symptoms of ectopic gestation may be perfectly simulated by other conditions. He describes the case of a patient with disturbed menstruation, bleeding, discharge of clots, pain, and recurrences; a soft, elastic swelling was found in Douglas' pouch, extending into the left fornix. The case seemed typical of ectopic gestation. At operation the swelling turned out to be a septic ovary, composed of a corpus luteum cyst, into which hemorrhage had occurred. The left tube was inflamed and contained pus; the right tube inflamed without suppuration.

In a paper entitled "Three Recent Cases of Tubal Pregnancy," Purslow (*Brit. Med. Jour.*, April 17, 1909) emphasizes the fact that in more than 50 per cent. of cases amenorrhœa does not precede the development of ectopic gestation. He considers the cardinal symptoms of this condition to be irregular and unusual vaginal hemorrhage, intermittent attacks of pain in the lower abdomen, or the presence of a tender swelling in the pelvis.

Adrenalin in the Treatment of Osteomalacia.—ENGLÄNDER (*Zentralbl. f. Gyn.*, 1909, No. 13) reports 6 cases of osteomalacia treated by the injection of adrenalin. Solution from 1 to 1000 was employed; injections were made daily, in the forenoon under strict antiseptic precautions, in the subcutaneous tissue of the abdominal wall. The temperature and pulse of the patient and her blood tension were observed and recorded daily. The doses varied from 0.5 to 1 c.c. Although the injections were somewhat painful, no local reaction followed. Some of the patients showed decided reaction in the redness and injection of the face, with altered conditions in the pupil. Most of the patients received 40 or more injections. Considerable improvement followed this treatment, the symptoms becoming less pronounced, and the patient being better able to do her work.

The Treatment of Placenta Prævia.—KRÖNIG (*Zentralbl. f. Gyn.*, 1909, No. 15) reports twenty cases of placenta prævia treated by combined and internal version, and contrasts the results obtained by this method with those following the treatment of placenta prævia by the use of a dilating bag. The significant feature in these cases is the fact that, without exception, version followed by bring down the body of the child stopped hemorrhage. This control of bleeding pertained during labor only, while the child was in the process of extraction, its body remaining within the genital canal of the mother. After the expulsion of the child, in most cases, hemorrhage of varying degree of severity occurred. Créde's method in many cases was inefficient in producing the discharge of the placenta, and it was necessary to empty the uterus by the introduction of the hand. The situation of the patient was critical for an hour or two after the delivery of the placenta. This postpartum bleeding is most severe in cases farthest advanced in pregnancy.

While these cases naturally develop frequently among patients in

their own homes, if the patient is carefully watched there is always some warning before severe hemorrhage begins. In 34 cases carefully investigated, there was preliminary hemorrhage sufficiently severe to attract attention in all of them. Such warning should lead to the patient's transfer to a hospital as soon as possible. When cases are seen in the very early stages of labor in an aseptic condition, and mother and child are in good condition, Cesarean section in a hospital will undoubtedly give the best result. The prognosis of placenta prævia is becoming very much better, the maternal mortality dropping to 6 per cent., and lower in cases promptly treated.

Spontaneous Rupture of the Umbilical Cord during Labor.—UNTERBERGER (*Zentralbl. f. Gyn.*, 1909, No. 14) reports two cases of spontaneous rupture of the umbilical cord during labor. In the first case the mother was a multipara, having a somewhat contracted pelvis. Labor pains were especially strong, the child being spontaneously expelled in the second position. The cord was about the neck, and the shoulders and body of the child were rapidly forced out after the birth of the head. The umbilical cord ruptured 22 cm. from the child's umbilicus, and the ends of the cord were immediately tied. The placenta was delivered spontaneously a little afterward. There was no hemorrhage from the uterus or from the cord. There was no laceration. The child was vigorous; the placenta contained many earthy deposits and a small infarct. There was no sign of syphilis. The cord was 47 cm. long. The second case was that of a primipara with slightly contracted pelvis, the child being expelled spontaneously with strong labor pains. The cord was also about the neck, and an effort to dislodge it was unsuccessful. The shoulders were born by a strong uterine contraction, and the cord ruptured 10 cm. from the child's umbilicus. The amnion sheath of the cord tore first, then both arteries, and finally the vein, the rupture of the cord permitting the expulsion of the shoulders. The tear across the cord was oblique with jagged edges. The ends were immediately tied, and hemorrhage did not occur. The cord was 50 cm. long, spiral, and with a great quantity of Wharton's jelly.

The Expulsion of the Uterine Decidua in Tubal Pregnancy.—BUCHÉ (*Zentralbl. f. Gyn.*, 1909, No. 13) reports the case of a multipara who had complained for four weeks of pain in the back and right lower abdomen, and had had four attacks of sharp pain in this vicinity. On examination a tumor the size of a goose-egg could be detected at the right side of the uterus. Douglas' cul-de-sac was filled by the tumor. At operation tubal pregnancy on the right side was found, with beginning hematocele surrounding the tube. On separating the tube the ovum was expelled into the abdominal cavity and removed practically entire. The uterine decidua was expelled in a complete cast of the uterus on the second day after operation. The shape of the sac was three-cornered, with prolongations which had evidently extended into the Fallopian tube. There were three openings to the sac, one to the cervix, the others at each tubal extremity. The lateral portions of the sac were thin and anemic, the middle more red and richly supplied with blood. Buché distinguishes three different ways in which the uterine

decidua is usually expelled: First, when the portions of the decidua in the tubes separate with difficulty, the mass is apt to come away entire. If this not the case, the decidua may be anteverted, the portion from the fundus being delivered first; or it may be delivered in situ, when the fundal decidua and that in the right uterine segment are loosened at the same time.

GYNECOLOGY.

UNDER THE CHARGE OF

J. WESLEY BOVÉE, M.D.,

PROFESSOR OF GYNECOLOGY IN THE GEORGE WASHINGTON UNIVERSITY, WASHINGTON, D. C.

Primary Carcinoma of the Female Urethra.—L. S. McMURTRY (*Trans. Amer. Surg. Assoc.*, 1908) reports two cases of primary carcinoma of the female urethra. He refers to the paucity of literature on the subject, and states that Percy's investigation of the 28 cases collected by Wassermann shows that but 3 of them were undoubtedly cancer developing primarily in the urethra. But Percy tabulated 16 genuine cases, including his one, that have been reported. The first was by Madame Boivin, in 1828, and the last in 1902. McMurtry adds 10 new ones, including his 2, that have since occurred. Caruncle is so similar to the early stage of cancer of the urethra that they are confounded. McMurtry advises very early excision.

Cyst of the Vagina.—ABADIE and RANGE (*Ann. de gyn. et d'obst.*, 1909, xxxvi, 71) report two cases of cyst of the vagina, and hold to the opinion that such cysts are developed from the Wolffian ducts. In the first case the patient was thirty-eight years of age, and the cyst, the size of a walnut, was located in the roof of the vagina on the under surface of the right broad ligament. It was successfully removed by one operation. The second specimen was removed from a multipara aged twenty-two years. The tumor was in the right vaginal wall and developed into the labium majus, and had attained a size about equal to that of the first case. The microscopic findings in these two cases furnish a basis for argument favorable to the congenital origin of these cysts—that is, their origin from the ducts of Gaertner.

Posterior Sagittal Incision of the Cervix Uteri.—L. HERZL (*Zeit. f. Geb. u. Gyn.*, 1908, lxii, 465) describes his technique for posterior sagittal incision of the cervix uteri and recommends this operation in cases of stenosis of the cervical canal when the latter causes dysmenorrhœa and sterility, and particularly in acute anteflexions of the uterus. The operation is contra-indicated in any case in which disease of the uterus and surrounding tissues exists which may in itself serve as a causal factor in the production of dysmenorrhœa and sterility. This operation was employed at the Vienna General Polyclinic only after all conservative measures had been tried and had failed. Twenty-seven cases were re-

examined. Complete cure had been obtained in 16, partial cure in 8, and total failure in 2. Herzl draws in addition to his own on Lott's private practice, and reports cure of sterility after this operation in 6 out of 40 cases operated upon.

A New Technique in Perineorrhaphy.—CHARLES JEWETT (*Long Island Med. Jour.*, August, 1908) modifies G. R. Holden's perineorrhaphy, employing it in nearly all forms of incomplete lacerations of the perineum. The modification consists of a few figure-of-eight interrupted sutures placed in the vaginal wall, including the levator ani on both sides, and a few passed deeply through the skin near the vertical wound. Chromicized catgut is used for sutures.

The Treatment of Gonorrhœa in the Female.—SAMUELS (*New York Med. Jour.*, March 20, 1909) says the important points in the treatment of gonorrhœa in women are cleanliness, intra-urethral injections, and constant observation of details. No active treatment during the early stages of the disease is advised. After the acute stage has passed, daily intra-urethral injections of 1 per cent. solution of protargol are to be used and the strength of the solution gradually increased. When the gonococci have disappeared, this remedy is stopped and a mild solution of sulphate of zinc substituted. In the chronic cases Samuels advises local applications of nitrate of silver, 20 grains to the ounce. His conclusions are that: (1) All cases with a history of burning and scalding urination should be thoroughly examined for an existing urethritis. (2) If a urethritis is found, presume it is of a gonorrhœal origin if no foreign body is present. (3) Institute a thorough treatment in all cases, for if a cure cannot be effected complications may be prevented. (4) Employ the vaginal douche only after all traces of the primary infection have disappeared and never in the early stages.

Extensive Lacerations of the Cervix Uteri, Involving the Parametrium: Their Significance and Treatment.—GEORGE TUCKER HARRISON (*Virginia Med. Semi-Monthly*, 1909, xiv, 30) regards lacerations that include the cervix uteri and parametrium, extending in many instances through the peritoneum, as of great importance, primarily from the danger of infection of deep extraperitoneal structures and peritoneum, and secondarily from the troublesome cicatrices resulting. In the production of these extensive lesions Harrison believes the Bossi dilator will in the future play an important role. In the treatment of the severe hemorrhage that often attends such severe laceration Harrison recommends the double compression plan of Fritsch, the details of which are: The placenta is, first of all, removed; this is easily done, as the inner os uteri is either wide or lacerated. The uterus now contracts readily. The physician then, with the right hand, presses the anteflexed uterus as deep as possible into the pelvis, and in that way all coagula are forced out. The operator, standing on the left side of the bed, seizes the vulva with the left hand, pressing the *labia majora* together, and forces them above into the angle of the arch of the pubes, as if he would shove the entire pelvic floor into the pelvic cavity above. Now, with the hand above coöperating with the hand below, the parts are forcibly compressed. The lower arm is not so readily paralyzed, as its elbow rests

on the bed; when the upper arm is paralyzed, an assistant is instructed to press on the upper hand with his or her two hands—to lean on it, as it were. It matters not whether the laceration is on the one side or the other, pressure is made directly downward. The hands are held in this way for half an hour, three-quarters, or even longer. A bag of sand or shot may substitute the upper hand. Harrison claims for it the following advantages: (1) The entire treatment is extravaginal; danger of infection by the procedure is consequently excluded. (2) The blood also, which would be certainly lost with suture or tampon, remains in the body. (3) The hemorrhage is controlled in the most rational manner from the fact that the bleeding surfaces are pressed together so firmly that there is no longer a space in which bleeding could take place. Provision is consequently made for adhesion and healing from the fact that the raw surfaces are on and into each other. (4) No after-treatment is necessary; when the hands are released, if no bleeding takes place, further manipulation is superfluous.

The Best Fixation Method for the Treatment of Fixed Retroversion with Flexion.—KUESTNER (*Zentralbl. f. Gyn.*, 1909, xxxiii, 41) states that reëxamination of the material at his clinic by Hannes showed that the Alexander-Adams operation gave most excellent results in those cases only in which the uterus was found freely movable. The Alexander-Adams operation should not be undertaken when any doubt of mobility of the uterus exists. Kuestner recommends the combination of the Alexander-Adams operation with laparotomy when adhesions are present. The former may be done unilaterally if desired. In cases in which careful examination fails to establish positively the presence of adhesions a suprasymphyseal transverse incision through the skin between the two external inguinal rings down to the fascia is made. The fascia is then divided longitudinally within the linea alba and the peritoneum opened. If no adhesions are found on exploratory incision of this kind one is justified in concluding the operation by the Alexander-Adams method.

Suprapubic Hysterotomy as a Means of Diagnosis and Treatment of the Uterus.—W. W. RUSSELL (*Ann. Gyn. and Ped.*, 1909, xxii, 90) advocates suprapubic hysterotomy for the following conditions: (1) It is of conspicuous service in cases of persistent uterine hemorrhage which is not controlled by any form of treatment and in which bimanual palpation of the pelvic organs has given an absolutely negative diagnosis. (2) In polypoid change of the mucosa in which frequent curettage has failed to stop the hemorrhage. (3) Small, submucous and pediculated myomas may be removed by this operation. (4) In cases in which the abdomen has been opened for some other purpose, and no explanation has been found. Russell reports 32 successful cases in which this procedure has been used by six surgeons. The technique consists of bringing the uterus out of the peritoneal cavity and, supported by gauze packed about it, a longitudinal incision into the uterus is made in the median line on the anterior wall half way down to the cervix, as well as through the fundus to the posterior surface. The entire uterus is split for a distance of two or more centimeters down into the cavity. In closing the uterine wall catgut or fine silk is placed through it about 0.5 cm. apart and a fine catgut closes the peritoneum.

DERMATOLOGY.

UNDER THE CHARGE OF
LOUIS A. DUHRING, M.D.,
PROFESSOR OF DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA,
AND
MILTON B. HARTZELL, M.D.,
ASSOCIATE IN DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

Lichen Ruber Planus as a Family Affection.—VEIEL (*Archiv f. Dermat. und Syph.*, 1909, xciii, Heft 3) has observed a family of four members in which three, the father and two children, suffered from typical lichen ruber planus, the father and one daughter several times. Veiel concludes from this observation and a number of similar ones in literature that there are without doubt families in which a certain hereditary predisposition to lichen ruber planus may be demonstrated, the term "disposition" being here employed in its widest sense.

Septicemia and Chronic Pemphigus Due to Bacillus Pyocyaneus.—PETGES and BICHELONNE (*Ann. de dermat. et de syphil.*, 1908, No. 7) report a striking example of chronic pemphigus associated with septicemia due to infection by Bacillus pyocyaneus, it being the first case reported, according to the authors, in which this organism has been found in the blood of patients with this affection. The patient was a man, aged twenty-two years, who, during a period of nine months, suffered from a bullous eruption presenting all the features of pemphigus which terminated fatally with symptoms of septicemia. At various times the urine presented a bluish-green hue; and from abscesses which formed on the scalp, the thigh, and in the axilla blue pus was evacuated. Cultures made from the blood antemortem and postmortem revealed the presence of Bacillus pyocyaneus. Petges and Bichelonne conclude that Bacillus pyocyaneus may be pathogenic for man, producing general diseases with or without cutaneous lesions, and diseases of the skin of apparently external origin. It may play a role in the production of chronic bullous pemphigus and pemphigus vegetans. The case which they reported they regard as incontestably caused by pyocyanic septicemia.

Biological Investigations in Pemphigus Vulgaris.—BRUCK (*Archiv f. Dermat. und Syph.*, 1909, Band xciii, Heft 3) who has recently employed biological methods in the study of two cases of pemphigus, having in mind the possibility of the presence of antigen in the fluid of the pemphigus blebs together with an antibody in the blood, tested this fluid with blood serum, but the results were completely negative. He found, however, that the fluid from the blebs of one of the patients contained a lysin which acted fairly strongly with human blood. Although the fluid was found sterile it contained a strong streptococcic virus. Inoculation of this sterile fluid into the skin of a pemphigus patient who had been free from lesions for fourteen days, produced after

some hours a hazelnut-sized bleb filled with a clear serous fluid, and in the course of the same and the next day a moderate number of blebs appeared on various parts of the body, which disappeared entirely in the next three days. A repetition of this experiment five days later was followed by a similar result. Bruck finds from this study that bacterial toxins may be demonstrated in the fluid of the bullæ present in pemphigus patients. He suggests the probable usefulness of antistreptococcic serum in the treatment of such cases as are accompanied by streptococcic infection, and thinks it might be worth while to attempt to influence the thermolabile toxins deposited in the skin through hot-air treatment.

Crude Coal Tar in the Treatment of Eczema.—Brocq (*Bull. de la soc. Francaise de dermat. et de syphil.*, 1908, No. 9) reports a number of patients with eczema who had been successfully treated with crude coal tar, which he regards as superior as a siccative and antipruritic to all other topical applications. He employs this agent in the following manner: The surface to be treated is first thoroughly freed from crusts and scales and then carefully washed with boiled water, and if not too sensitive it is afterward soaped and wiped off with ether. The tar is then applied in a thick layer and allowed to dry for a considerable time, the longer the better, the drying being an important part of the technique. When it has dried as long as possible, not less than twenty minutes, several hours if possible, it is powdered with talc and enveloped in a soft cloth. If the skin is not too much inflamed nor the oozing too abundant, it is well not to touch the dressing for two days. If the inflammation and oozing are marked it is useful to dress the parts with a simple zinc paste the next day. After five to six days the application of the tar may be repeated. Usually three to five applications are sufficient for a cure. Exceptionally it may excite inflammation, but Brocq finds that it is better tolerated than almost any other local remedy.

The Diagnostic and Specific Significance of the von Pirquet Skin Reaction.—Meirowsky (*Archiv f. Derm. u. Syphilis*, 1909, xciv, Heft 2 und 3) finds that a large percentage of those who present none of the clinical signs of tuberculosis give a positive cutaneous reaction with undiluted A. T. A positive result is also obtained when the subject has had a tuberculosis and any dermatosis; in such cases the cutaneous reaction is to be referred to the preceding tuberculous infection and not to the skin affection. Of 105 cases of tuberculosis of the skin, 102 gave a positive cutaneous reaction with undiluted A. T. Of the three which showed no reaction, two had been given simultaneously a subcutaneous injection of tuberculin; and after the resultant reaction had subsided both of these gave a positive cutaneous reaction. In only one case of lupus was the cutaneous reaction permanently negative. Meirowsky concludes that almost all cases of tuberculosis of the skin give a positive cutaneous reaction; and he believes that in a patient with a doubtful dermatosis a negative result makes it highly probable that the affection is non-tuberculous. He also thinks that the fact that the cutaneous reaction runs parallel with the reaction of the organism after injection of old tuberculin, proves the specificity of this reaction.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
WARFIELD T. LONGCOPE, M.D.,
DIRECTOR OF THE AYER CLINICAL LABORATORY, PENNSYLVANIA HOSPITAL.
ASSISTED BY
G. CANBY ROBINSON, M.D.,
EX-RESIDENT PHYSICIAN, PENNSYLVANIA HOSPITAL, PHILADELPHIA.

The Influence upon Intestinal Absorption of Exclusion of the Pancreatic Juice from the Intestines.—After discussing the results obtained by various observers upon the fat and nitrogen absorption by ligating the pancreatic duct in dogs, NIEMANN (*Ztschr. f. exp. Path. und Therap.*, 1909, xv, 466) describes two similar experiments which he performed. The main pancreatic duct together with the accessory duct was ligated, the dogs were allowed to recover, and later feeding experiments made. In both cases it was found that from 61.5 to 93.9 per cent. of the nitrogen of the food was absorbed, and from 91.2 to 98.7 per cent. of the fat. This seemed conclusive evidence that complete occlusion of the pancreatic ducts is compatible with excellent resorption of fat and proteid from the intestines. The effect on the pancreas of this operation is, however, very marked. When the dogs were killed, one or two months after operation, almost no normal pancreatic tissue was to be found. There occurs shortly after ligation of the ducts necrosis, first, of the central portion of the gland, and later, of the periphery. As the process extends toward the periphery the necrotic tissue is replaced by connective tissue.

Sclerosis of the Gastric Arteries.—The study of cases of arteriosclerosis in which the arteries supplying the stomach and intestines have been especially involved has led to the recognition of a more or less definite symptom complex spoken of as "Angina Abdominis." Perutz and Cheinisse have recently discussed this interesting question and have reviewed the literature extensively. The cases may be divided into three groups: (1) Those in which there are subjective and objective symptoms, pain and constipation during life, accompanied at autopsy by sclerosis of the abdominal aorta with thrombosis or stenosis of the opening of the cœliac axis or mesenteric arteries; (2) cases in which the same symptoms occur, with no other alterations than extensive sclerosis of the main branches or even finer twigs of the gastric and mesenteric arteries, and (3) those cases in which the disease is localized and manifests itself by ulceration of the gastro-intestinal mucous membrane or by hemorrhages. BUDAY (*Ziegler's Beit.*, 1908, xliv, 327) reports a case belonging to this third group. A man, aged forty-six years, had had, during the space of three years, several gastric hemorrhages. The last hemorrhage occurred five to six days before his illness, which was due apparently to collapse consequent upon the severe anemia. At autopsy old blood was found in the lower part of

the intestinal tract, but none was seen either in the duodenum or stomach. A careful examination of the entire gastro-intestinal tract failed to reveal any point of erosion or ulceration from which the bleeding might have arisen. There were, further, no changes in any of the other organs which could in any way explain the gastric hemorrhage. When, however, the gastric mucosa was searched again for possible bleeding points, it was found that the arteries in the wall of the stomach were the seat of an extreme grade of sclerosis, and in places groups of minute tortuous arteries could be felt as hard knots beneath the mucosa. Though there was a slight general sclerosis, there was in no organ, available for microscopic study, a sclerosis to compare with that of the gastric vessels. The arteries supplying the intestines were only moderately affected. Microscopic study of the gastric mucosa confirmed the findings at autopsy. The small arteries lying in the submucosa showed extensive alterations. There was great degeneration, sometimes calcification, and always thinning of the media with irregular dilatation of the vessel and thickening of the intima. The lumen of the vessels was rarely narrowed and never occluded. More often there was an aneurysmal dilatation. In places these diffuse aneurysms lay directly beneath the mucosa, and often the mucosa was much thinned above them. Buday concludes that the severe gastric hemorrhages in this case were due to rupture of these sclerotic vessels. In no place could an actual rupture be demonstrated, but since the last hemorrhage occurred over two weeks before death, and as there was no fresh blood in the stomach, it is fair to suppose that if a rupture had taken place it would have healed. Though Gallord, Sachs, and Hirschfeld have reported gastric hemorrhages from erosions of small arteries, very little attention has been called to this origin of the so-called idiopathic hemorrhages from the stomach, and Buday believes that a careful study of the arteries in such instances will lead to the finding of alterations such as he describes.

Experimental Heart Hypertrophy.—Following injections of adrenalin in rabbits, hypertrophy of the heart has been noted by certain observers, but so far the reason for this hypertrophy has not been satisfactorily explained. MIESOVIEZ (*Wiener klin. Woch.*, 1909, xxii, 79) has compared the weights of the hearts of normal rabbits with the weights of the hearts from rabbits which had received repeated injections of adrenalin. Since the actual weights of the heart of rabbits varied within wide limits it was only possible to use the relative weight of the heart to the body weight. This figure was fairly constant. In all the rabbits inoculated with adrenalin, hypertrophy of the heart was found, and the hypertrophy affected almost exclusively the left ventricle. The importance of sclerosis of the splanchnic arteries in the causation of heart hypertrophy in man, as pointed out by Romberg and Hasenfeld, suggested that the change in the bloodvessels caused by the adrenalin injections might account for the cardiac hypertrophy; but on analysis it was found that in several cases in which there was well-marked hypertrophy arterial changes were entirely absent. It was also found that there was no constant relationship between the numbers of injections of adrenalin and the amount of cardiac hypertrophy; and well-marked hypertrophy occurred after comparatively few injections.

Bearing upon this question are the observations of LOEB and FLEISCHER (*Jour. Exp. Med.*, 1909, x) who found that there occurs regularly in rabbits after the injection of adrenalin, myocarditic lesions of the left ventricle. These lesions can be seen with the naked eye, occur early, and appear to be more constant and characteristic for the injections of adrenalin than the arterial lesions. They consist in an hypertrophy of the muscle fibers with increase in the number of muscle nuclei, and indistinctness of cross striæ, increase in the connective tissue, which occurs early, and degenerative processes affecting the muscle fibers which become especially marked at a somewhat later stage. The authors call attention to the analogy which exists between these lesions and the changes described in hypertrophied hearts in man.

The Multiplication of Diseased Lymph Nodes.—RUDOLPH HAMMERSCHLAG (*Virchow's Arch.*, 1908, cxciv, 320) studied, principally by means of serial sections, the form and arrangement of packets or groups of enlarged lymph nodes from such condition as tuberculosis, sarcoma, and carcinoma, and points out two methods, which, so far, seem to have escaped notice by which multiplication of lymph nodes may take place. He has noticed that many of the large lymph nodes are composed really of two or more lymphoid masses, separated, sometimes partially, sometimes almost completely, by a definite zone of connective tissue. These separate lymphoid masses may give the node an irregular, lobulated appearance. Such lobules arise from sprouts which grow from the original node and are gradually cut off by a band of connective tissue growing from the hilum of the node. These sprouts may become sessile and finally appear as new glands lying next to the original node. In another group of cases the actual number of lymph nodes is increased by the proliferation of the lymph follicles in the cortex of a node. The proliferated follicles become separated from the original node and form new glands.

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DR. A. O. J. KELLY, 1911 Pine Street, Philadelphia, U. S. A

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